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* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 3	OCT 19	BEILSTEIN updated with new compounds
NEWS 4	NOV 15	Derwent Indian patent publication number format enhanced
NEWS 5	NOV 19	WPIX enhanced with XML display format
NEWS 6	NOV 30	ICSD reloaded with enhancements
NEWS 7	DEC 04	LINPADOCDB now available on STN
NEWS 8	DEC 14	BEILSTEIN pricing structure to change
NEWS 9	DEC 17	USPATOLD added to additional database clusters
NEWS 10	DEC 17	IMSDRUGCONF removed from database clusters and STN
NEWS 11	DEC 17	DGENE now includes more than 10 million sequences
NEWS 12	DEC 17	TOXCENTER enhanced with 2008 MeSH vocabulary in MEDLINE segment
NEWS 13	DEC 17	MEDLINE and LMEDLINE updated with 2008 MeSH vocabulary
NEWS 14	DEC 17	CA/CAplus enhanced with new custom IPC display formats
NEWS 15	DEC 17	STN Viewer enhanced with full-text patent content from USPATOLD
NEWS 16	JAN 02	STN pricing information for 2008 now available
NEWS 17	JAN 16	CAS patent coverage enhanced to include exemplified prophetic substances
NEWS 18	JAN 28	USPATFULL, USPAT2, and USPATOLD enhanced with new custom IPC display formats
NEWS 19	JAN 28	MARPAT searching enhanced
NEWS 20	JAN 28	USGENE now provides USPTO sequence data within 3 days of publication
NEWS 21	JAN 28	TOXCENTER enhanced with reloaded MEDLINE segment
NEWS 22	JAN 28	MEDLINE and LMEDLINE reloaded with enhancements
NEWS 23	FEB 08	STN Express, Version 8.3, now available
NEWS 24	FEB 20	PCI now available as a replacement to DPCI
NEWS 25	FEB 25	IFIREF reloaded with enhancements
NEWS 26	FEB 25	IMSPRODUCT reloaded with enhancements
NEWS 27	FEB 29	WPINDEX/WPIDS/WPIX enhanced with ECLA and current U.S. National Patent Classification

NEWS EXPRESS FEBRUARY 08 CURRENT WINDOWS VERSION IS V8.3,
AND CURRENT DISCOVER FILE IS DATED 20 FEBRUARY 2008

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 10:04:14 ON 03 MAR 2008

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.42	0.42

FILE 'REGISTRY' ENTERED AT 10:05:08 ON 03 MAR 2008
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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 2 MAR 2008 HIGHEST RN 1006303-40-7
DICTIONARY FILE UPDATES: 2 MAR 2008 HIGHEST RN 1006303-40-7

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TSCA INFORMATION NOW CURRENT THROUGH January 9, 2008.

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REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

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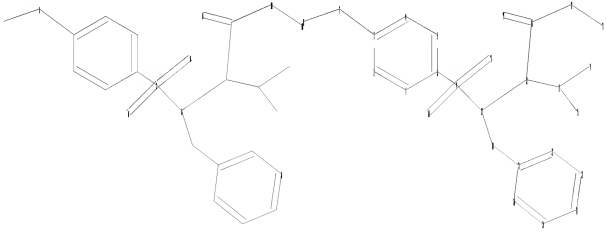
Uploading C:\Program Files\STNEXP\Queries\10506936.str



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chain nodes :
7  8  9  10  11  12  13  14  15  16  17  18  19  20  27
ring nodes :
1  2  3  4  5  6  21  22  23  24  25  26
chain bonds :
3-7  6-9  7-8  9-10  9-11  9-27  12-16  12-13  12-27  13-14  13-15  16-19  16-17
17-18  20-21  20-27
ring bonds :
1-2  1-6  2-3  3-4  4-5  5-6  21-22  21-26  22-23  23-24  24-25  25-26
exact/norm bonds :
3-7  6-9  7-8  9-10  9-11  9-27  12-27  16-19  16-17  20-27
exact bonds :
12-16  12-13  13-14  13-15  17-18  20-21
normalized bonds :
1-2  1-6  2-3  3-4  4-5  5-6  21-22  21-26  22-23  23-24  24-25  25-26


Match level :
1:Atom  2:Atom  3:Atom  4:Atom  5:Atom  6:Atom  7:CLASS  8:CLASS  9:CLASS  10:CLASS
 11:CLASS  12:CLASS  13:CLASS  14:CLASS  15:CLASS  16:CLASS  17:CLASS  18:CLASS
19:CLASS  20:CLASS  21:Atom  22:Atom  23:Atom  24:Atom  25:Atom  26:Atom  27:CLASS



L1      STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1              STR
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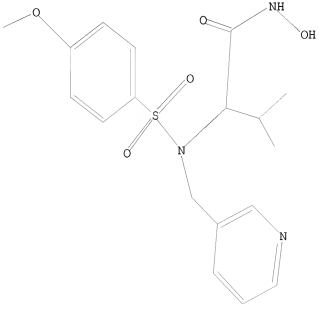


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Structure attributes must be viewed using STN Express query preparation.


=> s l1 sss ful
FULL SEARCH INITIATED 10:05:32 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -      172 TO ITERATE
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100.0% PROCESSED 172 ITERATIONS 24 ANSWERS
SEARCH TIME: 00.00.01

L2 24 SEA SSS FUL L1

=> s l1 fam ful
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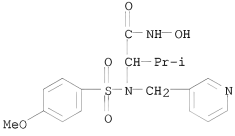
FULL SCREEN SEARCH COMPLETED - 66 TO ITERATE

100.0% PROCESSED 66 ITERATIONS 9 ANSWERS
SEARCH TIME: 00.00.01

L3 9 SEA FAM FUL L1

=> d scan l3

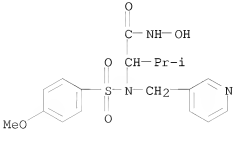
L3 9 ANSWERS REGISTRY COPYRIGHT 2008 ACS on STN
IN Butanamide, N-hydroxy-2-[[(4-methoxyphenyl)sulfonyl](3-pyridinylmethyl)amino]-3-methyl-
MF C18 H23 N3 O5 S
CI COM



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L3 9 ANSWERS REGISTRY COPYRIGHT 2008 ACS on STN
IN Butanamide, N-hydroxy-2-[[(4-methoxyphenyl)sulfonyl](3-pyridinylmethyl)amino]-3-methyl-, monohydrochloride (9CI)
MF C18 H23 N3 O5 S . Cl H



● HCl

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HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):end

=> file caplus
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                  ENTRY    SESSION
FULL ESTIMATED COST                               248.47     248.89

FILE 'CAPLUS' ENTERED AT 10:06:18 ON 03 MAR 2008
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)
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FILE COVERS 1907 - 3 Mar 2008  VOL 148 ISS 10
FILE LAST UPDATED: 2 Mar 2008  (20080302/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l3
L4          111 L3

=> s l4 and py<=2003
      23979372 PY<=2003
L5           72 L4 AND PY<=2003

=> s l5 and (cancer? or tumor? or neoplasm?)
         366619 CANCER?
         505848 TUMOR?
         502023 NEOPLASM?
L6           21 L5 AND (CANCER? OR TUMOR? OR NEOPLASM?)

=> d l6 ibib abs 1-21

L6   ANSWER 1 OF 21  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2004:166330  CAPLUS
DOCUMENT NUMBER:          141:17052
TITLE:                    Screening of stress enhancer based on analysis of gene
                          expression profiles: Enhancement of
                          hyperthermia-induced tumor necrosis by an
                          MMP-3 inhibitor
AUTHOR(S):                Kato, Naoki; Kobayashi, Takeshi; Honda, Hiroyuki
CORPORATE SOURCE:         Department of Biotechnology, School of Engineering,
                          Nagoya University, Nagoya, 464-8603, Japan
SOURCE:                   Cancer Science (2003), 94(7), 644-649
                          CODEN: CSACCM; ISSN: 1347-9032
PUBLISHER:                Japanese Cancer Association
DOCUMENT TYPE:            Journal
```

LANGUAGE: English

AB To improve the therapeutic benefit of hyperthermia, we examined changes of global gene expression after heat shock using DNA microarrays consisting of 12,814 clones. HeLa cells were treated for 1 h at 44° and RNA was extracted from the cells 0, 3, 6, and 12 h after heat shock. The 664 genes that were up or down-regulated after heat shock were classified into 7 clusters using fuzzy adaptive resonance theory (fuzzy ART). There were 41 genes in two clusters that were induced in the early phase after heat shock. In addition to shock response genes, such as hsp70 and hsp40, the stress response genes c-jun, c-fos and egr-1 were expressed in the early phase after heat shock. We also found that expression of matrix metalloproteinase 3 (MMP-3) was enhanced during the early response. We therefore investigated the role of MMP-3 in the heat shock response by examining HeLa cell survival after heat treatment in the presence and absence of an MMP-3 inhibitor, N-isobutyl-N-(4-methoxyphenyl-sulfonyl)glycylhydroxamic acid (NNGH) or N-hydroxy-2(R)-[[4-methoxysulfonyl](3-picolyl)amino]-3-methylbutaneamidehydrochloride(MMI270). The number of surviving cells 3 days after heat treatment significantly decreased, reaching 3.5% for NNGH and 0.2% for MMI270. These results indicate that the MMP-3 inhibitors enhanced heat shock-induced cell death and behaved as stress enhancers in cancer cells. This valuable conclusion was reached as a direct result of the gene expression profiling that was performed in these studies.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:855697 CAPLUS

DOCUMENT NUMBER: 139:364941

TITLE: Preparation of 3,4-diaminocyclobutene-1,2-diones as CXC chemokine receptor antagonists

INVENTOR(S): Taveras, Arthur G.; Aki, Cynthia J.; Bond, Richard W.; Chao, Jianping; Dwyer, Michael; Ferreira, Johan A.; Pachter, Jonathan A.; Baldwin, John J.; Kaiser, Bernd; Li, Ge; Merritt, J. Robert; Nelson, Kingsley H.; Rokosz, Laura L.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 127 pp., Cont.-in-part of U.S. Ser. No. 62,006.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2003204085	A1	20031030	US 2002-208426		20020730	<--
US 2003097004	A1	20030522	US 2002-62006		20020201	<--
US 2004235908	A1	20041125	US 2004-869189		20040616	
PRIORITY APPLN. INFO.:			US 2001-265951P	P	20010202	
			US 2002-62006	A2	20020201	
			US 2002-208426	A3	20020730	

OTHER SOURCE(S): MARPAT 139:364941

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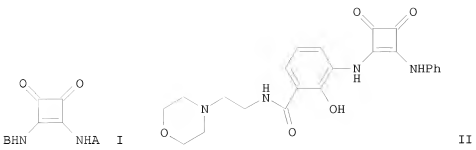

AB Title compds. I [A = (substituted) aryl, heteroaryl; B = (substituted) Ph, benzotriazolyl, benzimidazolyl, hydroxyimidazolyl, hydroxythienyl, hydroxypyrrolyl, etc.], useful for treating chemokine mediated diseases selected from psoriasis, atopic dermatitis, asthma, arthritis, cancer, etc., were prepared Thus, 1-ethoxy-2-phenylamino-1-cyclobutene-3,4-dione (preparation given) and 2-OH-3-[(2-morpholinoethyl)aminocarbonyl]aniline (preparation given) were refluxed overnight in EtOH to give 34% title compound (II). I showed CXCR2 receptor binding activity in the range of 1-10000 nM. Pharmaceutical composition comprising the compound I is claimed.

L6 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:786213 CAPLUS
DOCUMENT NUMBER: 140:296584
TITLE: Combination therapy of hepatocellular carcinoma with biological response modifiers
AUTHOR(S): Schuppan, D.; Herold, C.; Ganslmayer, M.; Ocker, M.; Hahn, E. G.
CORPORATE SOURCE: Innere Medizin I, Universitaet Erlangen-Nuernberg, Erlangen, D-91054, Germany
SOURCE: Malignant Liver Tumours: Basic Concepts and Clinical Management, Proceedings of [the] Falk Workshop, Leipzig, Germany, Jan. 24-25, 2002 (2003), Meeting Date 2002, 145-148. Editor(s): Berr, F. Kluwer Academic Publishers: Dordrecht, Neth. CODEN: 69EPH7; ISBN: 0-7923-8779-1
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review. The treatment of hepatocellular carcinoma (HCC) cells with tamoxifen (TAM), 9-cis-retinoic acid (CRA), TNP-470, or histone deacetylase inhibitors alone showed minor to moderate antiproliferative effects. Inhibitors of matrix metalloproteinases and of angiogenesis were ineffective. Modulation or inhibition of several tumor-specific alterations, such as neo-angiogenesis, peri-tumoral lysis of extracellular matrix and resistance to apoptosis, is a promising strategy for the treatment of solid gastrointestinal cancers, in particular HCC.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:757689 CAPLUS
DOCUMENT NUMBER: 139:276755
TITLE: Preparation of epothilone derivatives for therapeutic use as anticancer agents
INVENTOR(S): Regueiro-Ren, Alicia; Kim, Soong-Hoon
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 47 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003078411	A1	20030925	WO 2003-US7584	20030311 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003218110	A1	20030929	AU 2003-218110	20030311 <--
US 2003191089	A1	20031009	US 2003-386072	20030311 <--
US 6719540	B2	20040413		
EP 1483251	A1	20041208	EP 2003-714096	20030311
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: US 2002-363441P P 20020312
WO 2003-US7584 W 20030311

OTHER SOURCE(S): MARPAT 139:276755
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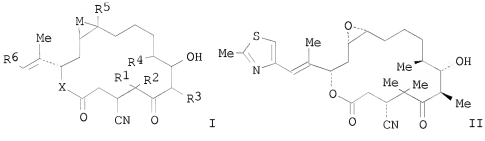

AB Epothilone derivs., such as I [M = bond, O, NR9, CR10R11; X = O, NH; R1-R4 = H, alkyl; R5 = H, alkyl, cyano; R6 = H, alkyl, aryl, heterocyclyl; R9-R11 = H, OH, alkyl, alkoxy, aryl, cycloalkyl, heterocyclyl], pharmaceutically acceptable salts, solvates or hydrate thereof, were prepared for use as antitumor agents. Thus, epothilone derivative II was prepared from 2,3-dehydro epothilone A, via silylation of hydroxyl group, potassium cyanide addition, followed by deprotection. The prepared epothilone derivs. were assayed in vitro for their effect on tubulin polymerization and for cytotoxicity against HCT-116 human colon carcinoma cells. Therapeutic compns. containing I or in combination with other therapeutic agents useful in the treatment of cancer or other proliferative diseases are also claimed.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:757513 CAPLUS
DOCUMENT NUMBER: 139:276754

TITLE: Preparation of C12-cyano epothilone derivatives with antitumor activity

INVENTOR(S): Vite, Gregory D.; Regueiro-Ren, Alicia

PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA

SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003077903	A1	20030925	WO 2003-US7576	20030311 <--
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003218107	A1	20030929	AU 2003-218107	20030311 <--
US 2003186965	A1	20031002	US 2003-386059	20030311 <--
US 7211593	B2	20070501		
PRIORITY APPLN. INFO.:			US 2002-363703P	P 20020312
			WO 2003-US7576	W 20030311

OTHER SOURCE(S): MARPAT 139:276754

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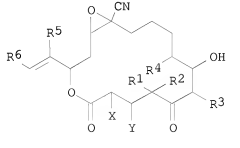

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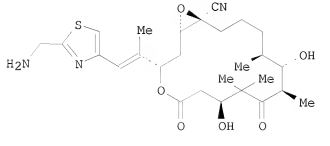

II

AB Epothilone derivs. of formula I [R1-R5 = H, alkyl; R6 = H, alkyl, aryl, cycloalkyl, heterocyclo; X = H; Y = OH; XY = bond] are prepared Also included are therapeutic compns. containing the compds. of formula I as active ingredients, alone or in combination with other therapeutic agents useful in the treatment of cancer or other proliferative diseases. Thus, II was prepared in several steps from epothilone A. The EC0.01 of the

prepared compds. was 0.01 to 1000 μM in in vitro tubulin polymerization assay.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS
DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307 <--
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RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307 <--
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606

PRIORITY APPLN. INFO.: GB 2002-5537 A 20020308
GB 2002-29054 A 20021212
WO 2003-EP2365 W 20030307

OTHER SOURCE(S): MARPAT 139:240351
AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

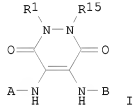
L6 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:551500 CAPLUS
DOCUMENT NUMBER: 139:117431
TITLE: 4,5-Diamino-1,2,3,4-tetrahydro-3,6-pyridazinediones as CXC chemokine receptor antagonists for treatment of inflammatory disorders and cancer
INVENTOR(S): Taveras, Arthur G.; Dwyer, Michael; Chao, Jianping; Baldwin, John J.; Merritt, Robert J.; Li, Ge; Chao, Jianhua; Yu, Younong
PATENT ASSIGNEE(S): Schering Corporation, USA; Pharmacopeia, Inc.
SOURCE: PCT Int. Appl., 210 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003057676	A1	20030717	WO 2003-US299	20030103 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SC, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VC, VN, YU, ZA, ZM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004063709	A1	20040401	US 2003-335789	20030102
US 6878709	B2	20050412		
CA 2472165	A1	20030717	CA 2003-2472165	20030103 <--
AU 2003207460	A1	20030724	AU 2003-207460	20030103 <--
EP 1461321	A1	20040929	EP 2003-705667	20030103
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1582280	A	20050216	CN 2003-801923	20030103
JP 2005516029	T	20050602	JP 2003-557993	20030103
MX 2004PA06555	A	20041004	MX 2004-PA6555	20040702

PRIORITY APPLN. INFO.:			
	US 2002-346248P	P	20020104
	US 2003-335789	A	20030102
	WO 2003-US299	W	20030103

OTHER SOURCE(S): MARPAT 139:117431
GI



AB Prepns. for title compds. I [wherein R1 and R15 = independently H or (un)substituted (hetero)aryl, alkyl, (hetero)cycloalkyl(alkyl), or (hetero)arylalkyl; A = (un)substituted thiazolyl(alkyl), thienyl(alkyl), oxazolyl(alkyl), pyridinyl(alkyl), piperazinyl(alkyl), piperidinyl(alkyl), imidazolyl(alkyl), indolyl(alkyl), benzotriazolyl(alkyl), phenyl(alkyl), naphthyl(alkyl), carbamoylalkyl, etc.; B = (un)substituted Ph, benzotriazolyl, benzimidazolyl, indolyl, indazolyl, pyridinyl, pyrazolyl, thienyl, pyrrolyl, or pyrimidinyl; or pharmaceutically acceptable salts or solvates thereof] and their intermediates are disclosed (no data). In addition, CXCR1 SPA, CXCR2 SPA, calcium fluorescence, chemotaxis, and cytotoxicity assays are described. For example, 5-methylsalicylic acid was coupled with dimethylamine in the presence of DCC in EtOAc to give 2-hydroxy-N,N,5-trimethylbenzamide, which was nitrated (44%) and reduced using 10% Pd/C to give 3-amino-2-hydroxy-N,N,5-trimethylbenzamide (99%). The amine may be coupled with 1,2,3,4-tetrahydro-3,6-pyridazinediones to provide compds. of the invention (no data). I may exhibit a range of CXCR2 receptor binding activities from about 1 nM to about 10,100 nM. Thus, I and pharmaceutical comps. comprising I may be useful for the treatment of acute and chronic inflammatory disorders and cancer

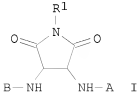
(no data).
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:301081 CAPLUS
DOCUMENT NUMBER: 138:321127
TITLE: Preparation of 3,4-disubstituted maleimide compounds as CXC-chemokine receptor antagonists
INVENTOR(S): Taveras, Arthur G.; Dwyer, Michael; Ferreira, Johan A.; Girijavallabhan, Viyyoor M.; Chao, Jianping; Baldwin, John J.; Merritt, J. Robert; Li, Ge
PATENT ASSIGNEE(S): Schering Corporation, USA; Pharmacopeia, Inc.
SOURCE: PCT Int. Appl., 229 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003031440	A1	20030417	WO 2002-US32628	20021011 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VC, VN, YU, ZA, ZM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2462862	A1	20030417	CA 2002-2462862	20021011 <--
AU 2002351478	A1	20030422	AU 2002-351478	20021011 <--
US 2004034229	A1	20040219	US 2002-269775	20021011
US 6903131	B2	20050607		
EP 1434775	A1	20040707	EP 2002-786395	20021011
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005505595	T	20050224	JP 2003-534423	20021011
CN 1599734	A	20050323	CN 2002-824052	20021011
MX 2004PA03439	A	20040708	MX 2004-PA3439	20040412

PRIORITY APPLN. INFO.:		US 2001-329005P	P 20011012
		WO 2002-US32628	W 20021011

OTHER SOURCE(S): MARPAT 138:321127
GI



AB Disclosed are 3,4-disubstituted maleimides (shown as I; variables defined below; e.g. 3-[[3-(dimethylcarbamoyl)-2-hydroxyphenyl]amino]-4-((tert-butyl)amino)maleimide) or pharmaceutically acceptable salts or solvates thereof. The compds. are useful for the treatment of chemokine-mediated

diseases such as acute and chronic inflammatory disorders and cancer. CXCR1 and CXCR2 SPA, calcium fluorescence, chemotaxis (for 293-CXCR2), cytotoxicity and soft agar receptor binding assay methods are described but no test results are reported. Although the methods of preparation are not claimed, 1 example preparation of I and a large number of example
prepns. of intermediates are included; also >200 specific I are claimed. For I: R1 = H or (un)substituted aryl, heteroaryl, alkyl, arylalkyl, heteroarylalkyl, cycloalkyl, heterocycloalkyl, cycloalkylalkyl, and heterocycloalkylalkyl; A is selected from a very large group of possibilities, e.g. CR7R8Z (Z = (un)substituted pyridinyl, 1-oxopyridinyl, thiazolyl, furyl, oxazolyl, imidazolyl); B is selected from a very large group of possibilities, e.g. (un)substituted Ph, benzotriazol-7-yl, thienyl; addnl. details are given in the claims.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:272967 CAPLUS
DOCUMENT NUMBER: 139:316768
TITLE: Anti-tumor angiogenic effect of a matrix metalloproteinase inhibitor MMI270
AUTHOR(S): Nakamura, Eliane Shizuka; Koizumi, Keiichi; Yamaura, Takeshi; Saiki, Ikuo
CORPORATE SOURCE: Department of Pathogenic Biochemistry, Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan
SOURCE: Anticancer Research (2003), 23(1A), 411-417
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We investigated the anti-angiogenic effects of a matrix metalloproteinase inhibitor, (MMI), so called MMI270, against B16-BL6 melanoma through the inhibition of the migrating and invasive abilities of hepatic sinusoidal endothelial (HSE) cells, as well as the formation of tube-like structures by HSE cells. MMI270, at the concentration of 12.5 μg/mL, significantly inhibited the migration and invasion of HSE cells, in addition to tube formation by approx. 40%. Furthermore, the enzymic degradation of metalloproteinases MMP-9 and MMP-2 produced by HSE cells was inhibited by treatment with 1 μg/mL of MMI270, showing 30% and 100% of inhibition in comparison to the control, resp. The i.p. administration of MMI270 (200 mg/kg, twice daily for 8 days) after the implantation of B16-BL6 melanoma cells into mice reduced the number of vessels towards the established primary tumor on the dorsal side of mice. These results suggest that MMI270 might be useful as an anti-tumor angiogenic drug.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:55812 CAPLUS
DOCUMENT NUMBER: 139:223633
TITLE: Role of body surface area in dosing of investigational anticancer agents in adults, 1991-2001
AUTHOR(S): Baker, Sharyn D.; Verweij, Jaap; Rowinsky, Eric K.; Donehower, Ross C.; Schellens, Jan H. M.; Grochow, Louise B.; Sparreboom, Alex
CORPORATE SOURCE: Division of Experimental Therapeutics, The Sydney Kimmel Comprehensive Cancer Center at Johns Hopkins, Baltimore, MD, USA
SOURCE: Journal of the National Cancer Institute (2002), 94(24), 1883-1888

CODEN: JNCIEQ; ISSN: 0027-8874
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The prescribed dose of anticancer agents is most commonly calculated using body surface area as the only independent variable, and it has been shown that this approach still results in large inter-patient variability in drug exposure. Here, we retrospectively assessed the pharmacokinetics of 33 investigational agents tested in phase I trials from 1991 through 2001, as a function of body surface area in 1650 adult cancer, patients. Twelve of the drugs were administered orally, 19 were administered i.v., and two were administered by both routes. Body surface area-based dosing was statistically significantly associated with a reduction in inter-patient variability in drug clearance for only five of the 33 agents: docosahexaenoic acid (DHA)-paclitaxel, 5-fluorouracil/eniluracil, paclitaxel, temozolomide, and troxacitabine. These results do not support the use of body surface area in dose calcns. and suggest that alternate dosing strategies should be evaluated. We conclude that body surface area should not be used to determine starting doses of investigational agents in future phase I studies.
REFERENCE COUNT: 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:868715 CAPLUS
DOCUMENT NUMBER: 137:346164
TITLE: Anti-angiogenic therapy using liposome-encapsulated chemotherapeutic agents
INVENTOR(S): Flowers, Clay; Saltman, David; Tam, Patrick M. S.; Burge, Clive T. R.; Harasym, Troy O.
PATENT ASSIGNEE(S): Inex Pharmaceuticals Corporation, Can.
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002089772	A1	20021114	WO 2002-US14608	20020509 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002256504	A1	20021118	AU 2002-256504	20020509 <--
US 2003082228	A1	20030501	US 2002-143545	20020509 <--
PRIORITY APPLN. INFO.:			US 2001-289935P	P 20010509
			WO 2002-US14608	W 20020509

AB The present invention provides methods and compns. for the treatment and prevention of any of a large number of diseases and conditions with an angiogenic component, e.g., cancer. The present invention is based upon the discovery that liposome-encapsulated chemotherapeutic agents, such as alkaloids (e.g., vinca alkaloids such as vincristine), are surprisingly effective at treating such diseases or conditions when administered at a higher frequency than those used with conventional

administration strategies. Such methods can be used to treat diseases such as cancer even when the cancer comprises cells that are resistant to the chemotherapeutic alkaloid. The liposome encapsulation of the chemotherapeutic agents, e.g., alkaloids, imparts dramatic improvements in the stability, biodistribution, and delivery of the agents, thereby allowing more efficacious and convenient administration to a patient with any of the herein-described diseases or conditions.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:754340 CAPLUS

DOCUMENT NUMBER: 137:279205

TITLE: Preparation of 3,4-diaminocyclobutene-1,2-diones as CXC chemokine receptor antagonists

INVENTOR(S): Taveras, Arthur G.; Aki, Cynthia J.; Bond, Richard W.; Chao, Jianping; Dwyer, Michael; Ferreira, Johan A.; Pachter, Jonathan; Baldwin, John J.; Kaiser, Bernd; Li, Ge; Merritt, J. Robert; Nelson, Kingsley H., Jr.; Rokosz, Laura L.

PATENT ASSIGNEE(S): Schering Corporation, USA; Pharmacopeia, Inc.

SOURCE: PCT Int. Appl., 113 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002076926	A1	20021003	WO 2002-US2888	20020201 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VN, YU, ZA, ZM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2436351	A1	20021003	CA 2002-2436351	20020201 <--
AU 2002303084	A1	20021008	AU 2002-303084	20020201 <--
EP 1355875	A1	20031029	EP 2002-731085	20020201 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2002006968	A	20040309	BR 2002-6968	20020201
HU 2003004047	A2	20040428	HU 2003-4047	20020201
JP 2004529911	T	20040930	JP 2002-576189	20020201
CN 1575273	A	20050202	CN 2002-804517	20020201
NZ 527947	A	20051028	NZ 2002-527947	20020201
IN 2003CN01171	A	20050422	IN 2003-CN1171	20030729
ZA 2003005881	A	20041101	ZA 2003-5881	20030730
NO 2003003424	A	20030930	NO 2003-3424	20030731 <--
MX 2003PA06950	A	20031118	MX 2003-PA6950	20030801 <--

PRIORITY APPLN. INFO.: US 2001-265951P P 20010202
WO 2002-US2888 W 20020201

OTHER SOURCE(S): CASREACT 137:279205; MARPAT 137:279205

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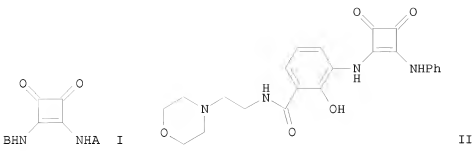

AB Title compds. I; [A = (substituted) aryl, heteroaryl; B = (substituted) Ph, benzotriazolyl, benzimidazolyl, hydroxyimidazolyl, hydroxythienyl, hydroxypyrrolyl, etc.], were prepared Thus, 1-ethoxy-2-phenylamino-1-cyclobutene-3,4-dione (preparation given) and 2-OH-3-[2-(morpholinoethyl)aminocarbonyl]aniline (preparation given) were refluxed overnight in EtOH to give 34% title compound (II). I showed CXCR2 receptor binding activity in the range of 1-10000 nM.

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:441843 CAPLUS

DOCUMENT NUMBER: 138:24507

TITLE: Synthesis of MMP inhibitor radiotracers [11C]methyl-CGS 27023A and its analogs, new potential PET breast cancer imaging agents

AUTHOR(S): Fei, Xiangshu; Zheng, Qi-Huang; Hutchins, Gary D.; Liu, Xuan; Stone, K. Lee; Carlson, Kathy A.; Mock, Bruce H.; Winkle, Wendy L.; Glick-Wilson, Barbara E.; Miller, Kathy D.; Fife, Rose S.; Sledge, George W.; Sun, Hui Bin; Carr, Raymond E.

CORPORATE SOURCE: Department of Radiology, Indiana University School of Medicine, Indianapolis, IN, 46202, USA

SOURCE: Journal of Labelled Compounds & Radiopharmaceuticals (2002), 45(6), 449-470
CODEN: JLCRD4; ISSN: 0362-4803

PUBLISHER: John Wiley & Sons Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 138:24507

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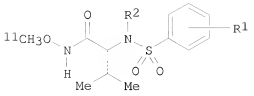

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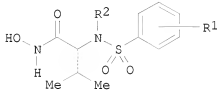

II

AB [11C]Methyl-CGS 27023A I [R1 = 4-MeO, R2 = (3-pyridinyl)methyl] and its analogs I [R1 = 4-MeO, R2 = (2-pyridinyl)methyl, PhCH2; R1 = 2-O2N, 3-O2N, 4-O2N, R2 = (3-pyridinyl)methyl], novel radiolabeled matrix metalloproteinase (MMP) inhibitors, were synthesized for evaluation as new potential positron emission tomog. (PET) breast cancer imaging agents. The precursors II, obtained in four to five steps from D-valine in moderate to excellent yields, were radiolabeled by methylation with [11C]methyl triflate at the aminohydroxyl position under basic conditions in 20-25 min synthesis time, and pure I were isolated by solid-phase extraction (SPE) purification in 40-60% radiochem. yields (decay corrected to end of bombardment).

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:359662 CAPLUS
DOCUMENT NUMBER: 137:306709
TITLE: Radiation-induced increase in invasive potential of human pancreatic cancer cells and its blockade by a matrix metalloproteinase inhibitor, CGS27023
AUTHOR(S): Qian, Li-Wu; Mizumoto, Kazuhiro; Urashima, Taro; Nagai, Eishi; Maehara, Naoki; Sato, Norihiro; Nakajima, Motowo; Tanaka, Masao
CORPORATE SOURCE: Department of Surgery and Oncology, Kyushu University, Fukuoka, 812-8582, Japan
SOURCE: Clinical Cancer Research (2002), 8(4), 1223-1227
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Purpose: Radiotherapy remains a major therapeutic option for patients with advanced pancreatic cancer. Nevertheless, the effects of irradiation on malignant biol. behaviors (e.g., migration and invasion of cancer cells) have yet to be clarified. Thus, we conducted an in vitro study to investigate the radiation-induced alterations around cell migration and invasion capacity. Experiment design: Three cell lines from human pancreatic cancer were included in the study. γ-Radiation was used for irradiation treatment. Cell migration and invasion ability were evaluated by Transwell migration assay and Matrigel invasion assay. The activity of MMP-2 and 9, and expression of

urokinase-type plasminogen activator were investigated with gelatin zymog. and immunoblot, resp. Results: Irradiation enhances invasive potential in some pancreatic cancer cells, whereas it significantly inhibits cell proliferation and migration. This hitherto unknown biol. effect of irradiation involves enhanced matrix metalloproteinase (MMP)-2 activity. Consequently, simultaneous administration of an MMP inhibitor, CGS27023A, suppresses the radiation-enhanced invasion through blockade of transition of MMP-2 from latent type to active type. Conclusion: Because radiation may increase invasion ability through activating MMP proteolytic system, simultaneous administration of the MMP inhibitor during radiotherapy could be a potent adjuvant therapeutic approach to improve the efficacy of radiotherapy for pancreatic cancer.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:935435 CAPLUS
DOCUMENT NUMBER: 136:84677
TITLE: Methods for enhancing antibody-induced cell lysis and treating cancer
INVENTOR(S): Weiner, George; Hartmann, Gunther
PATENT ASSIGNEE(S): University of Iowa Research Foundation, USA
SOURCE: PCT Int. Appl., 312 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001097843	A2	20011227	WO 2001-US20154	20010622 <--
WO 2001097843	A3	20030123		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2410371	A1	20011227	CA 2001-2410371	20010622 <--
AU 2001070134	A	20020102	AU 2001-70134	20010622 <--
US 2003026801	A1	20030206	US 2001-888326	20010622 <--
EP 1296714	A2	20030402	EP 2001-948684	20010622 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003535907	T	20031202	JP 2002-503327	20010622 <--
AU 2006216542	A1	20061012	AU 2006-216542	20060915
PRIORITY APPLN. INFO.:			US 2000-213346P	P 20000622
			AU 2001-270134	A3 20010622
			WO 2001-US20154	W 20010622

AB The invention relates to methods and products for treating cancer . In particular the invention relates to combinations of nucleic acids and antibodies for the treatment and prevention of cancer. The invention also relates to diagnostic methods for screening cancer cells.

L6 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:572678 CAPLUS
DOCUMENT NUMBER: 136:263

TITLE: Phase I and pharmacological study of the oral matrix metalloproteinase inhibitor, MMI270 (CGS27023A), in patients with advanced solid cancer

AUTHOR(S): Levitt, Nicola C.; Eskens, Ferry A. L. M.; O'Byrne, Ken J.; Propper, David J.; Denis, Louis J.; Owen, Samantha J.; Choi, Les; Foekens, John A.; Wilner, Sue; Wood, Jeanette M.; Nakajima, Motowo; Talbot, Denis C.; Steward, William P.; Harris, Adrian L.; Verweij, Jaap

CORPORATE SOURCE: Imperial Cancer Research Fund Unit, Churchill Hospital, Oxford, OX3 FLJ, UK

SOURCE: Clinical Cancer Research (2001), 7(7), 1912-1922
CODEN: CCREF4; ISSN: 1078-0432

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This Phase I study of MMI270, an p.o. administered matrix metalloproteinase inhibitor, assessed toxicity, pharmacokinetics, and tumor response data and investigated markers of biol. activity to recommend a dose for Phase II studies. MMI270 was administered continuously at seven dose levels (50 mg once daily to 600 mg three times/day). Patients were evaluated for toxicity and tumor response, and blood and urine samples were taken for pharmacokinetics, bone resorption markers, direct targets of the inhibitor [matrix metalloproteinase-2 (MMP-2), MMP-8, and MMP-9], indirect targets [tissue inhibitor of metalloproteinase-1 (TIMP-1), TIMP-2, basic fibroblast growth factor, vascular endothelial growth factor, vascular cell adhesion mol.-1, soluble urokinase plasminogen activator receptor, and cathepsins B and H] and for a tumor necrosis factor-α cytokine release assay. Ninety-two patients were entered. There was no myelo-toxicity. Eighteen patients developed a widespread maculopapular rash, which increased in frequency and severity at doses ≥300 mg bid. Thirty nine patients developed musculoskeletal side effects, which were related to duration of treatment, not to dose level. Pharmacokinetics were linear, and MMI270 was rapidly absorbed and eliminated with minimal accumulation on chronic dosing. Sustained plasma concns. in excess of 4 + mean IC50 for the target enzymes were observed at dose levels ≥150 mg bid. There were no tumor regressions; however, 19 patients had stable disease for ≥90 days. There was a dose-response increase of MMP-2 and TIMP-1 with MMI270. Transient effects on the bone resorption markers were detected. MMI270 was generally well tolerated, with adequate plasma levels for target enzyme inhibition. The two main toxicities were rash, resulting in a maximum tolerated dose of 300 mg bid and musculoskeletal side effects. Biol. marker data indicate drug effects. The rise in TIMP-1 suggests that a reflex rise in inhibitors could modify the effects of MMI270. The recommended Phase II dose is 300 mg bid.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:259436 CAPLUS

DOCUMENT NUMBER: 136:272422

TITLE: TACE and other ADAM proteases as targets for drug discovery

AUTHOR(S): Moss, M. L.; White, J. M.; Lambert, M. H.; Andrews, R. C.

CORPORATE SOURCE: Cognosci, Research Triangle Park, NC, 27709, USA

SOURCE: Drug Discovery Today (2001), 6(8), 417-426
CODEN: DDTOFS; ISSN: 1359-6446

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Tumor necrosis factor (TNF)-converting enzyme (TACE) and other ADAM proteases (those that contain a disintegrin and a metalloprotease domain) have emerged as potential therapeutic targets in the areas of arthritis, cancer, diabetes and HIV cachexia. TACE is the first ADAM protease to process the known physiol. substrate and inflammatory cytokine, membrane-bound precursor-TNF-α, to its mature soluble form. Subsequently, TACE was shown to be required for several different processing events such as tumor growth factor-α (TGF-α) precursor and amyloid precursor protein (APP) cleavage. With the recent discoveries of the proteolytic specificities of other ADAM family members, the information surrounding these metalloproteases is expanding at an exponential rate. This review focuses on TACE and other family members with known proteolytic function as well as the inhibitors of this class of enzyme.

REFERENCE COUNT: 104 THERE ARE 104 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1999:738391 CAPLUS

DOCUMENT NUMBER: 132:89906

TITLE: Catalytic activities and substrate specificity of the human membrane type 4 matrix metalloproteinase catalytic domain

AUTHOR(S): Wang, Yahong; Johnson, Adam R.; Ye, Qi-Zhuang; Dyer, Richard D.

CORPORATE SOURCE: Department of Biochemistry, Parke-Davis Pharmaceutical Research Division, Warner-Lambert Company, Ann Arbor, MI, 48105, USA

SOURCE: Journal of Biological Chemistry (1999), 274(46), 33043-33049
CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Membrane type (MT) matrix metalloproteinases (MMPs) are recently recognized members of the family of Zn2+- and Ca2+-dependent MMPs. To investigate the proteolytic capabilities of human MT4-MMP (i.e. MMP-17), we have cloned DNA encoding its catalytic domain (CD) from a breast carcinoma cDNA library. Human membrane type 4 MMP CD (MT4-MMPCD) protein, expressed as inclusion bodies in Escherichia coli, was purified to homogeneity and refolded in the presence of Zn2+ and Ca2+. While MT4-MMPCD cleaved synthetic MMP substrates Ac-PLG-[2-mercapto-4-methylpentanoyl]-LG-OEt and Mca-PLGL-Dpa-AR-NH2 with modest efficiency, it catalyzed with much higher efficiency the hydrolysis of a pro-tumor necrosis factor-α converting enzyme synthetic substrate, Mca-PLAQAV-Dpa-RSSSR-NH2. Catalytic efficiency with the pro-tumor necrosis factor-α converting enzyme substrate was maximal at pH 7.4 and was modulated by three ionizable enzyme groups (pKa3 = 6.2, pKa2 = 8.3, and pKa1 = 10.6). MT4-MMPCD cleaved gelatin but was inactive toward type I collagen, type IV collagen, fibronectin, and laminin. Like all known MT-MMPs, MT4-MMPCD was also able to activate 72-kDa progelatinase A to its 68-kDa form. EDTA, 1,10-phenanthroline, reference hydroxamic acid MMP inhibitors, tissue inhibitor of metalloproteinases-1, and tissue inhibitor of metalloproteinases-2 all potently blocked MT4-MMPCD enzymic activity. MT4-MMP is, therefore, a competent Zn2+-dependent MMP with unique specificity among synthetic substrates and the capability to both degrade gelatin and activate progelatinase A.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 19 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:103528 CAPLUS
DOCUMENT NUMBER: 130:293102
TITLE: Metalloprotease-disintegrin MDC9: intracellular maturation and catalytic activity
AUTHOR(S): Roghani, Monireh; Becherer, J. David; Moss, Marcia L.; Atherton, Ruth E.; Erdjument-Bromage, Hediye; Arribas, Joaquin; Blackburn, R. Kevin; Weskamp, Gisela; Tempst, Paul; Blobel, Carl P.
CORPORATE SOURCE: Cellular Biochemistry and Biophysics Program, Sloan-Kettering Institute Memorial Solan-Kettering Cancer Center, Graduate School of the Cornell University Medical College, New York, NY, 10021, USA
SOURCE: Journal of Biological Chemistry (1999), 274(6), 3531-3540
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Metalloprotease disintegrins are a family of membrane-anchored glycoproteins that are known to function in fertilization, myoblast fusion, neurogenesis, and ectodomain shedding of tumor necrosis factor (TNF)-α. Here we report the anal. of the intracellular maturation and catalytic activity of the widely expressed metalloprotease disintegrin MDC9. Our results suggest that the pro-domain of MDC9 is removed by a furin-type pro-protein convertase in the secretory pathway before the protein emerges on the cell surface. The soluble metalloprotease domain of MDC9 cleaves the insulin B-chain, a generic protease substrate, providing the first evidence that MDC9 is catalytically active. Soluble MDC9 appears to have distinct specificities for cleaving candidate substrate peptides compared with the TNF-α convertase (TACE/ADAM17). The catalytic activity of MDC9 can be inhibited by hydroxamic acid-type metalloprotease inhibitors in the low nanomolar range, in one case with up to 50-fold selectivity for MDC9 vs. TACE. Peptides mimicking the predicted cysteine-switch region of MDC9 or TACE inhibit both enzymes in the low micromolar range, providing exptl. evidence for regulation of metalloprotease disintegrins via a cysteine-switch mechanism. Finally, MDC9 shown to become phosphorylated when cells are treated with the phorbol ester phorbol 12-myristate 13-acetate, a known inducer of protein ectodomain shedding. This work implies that removal of the inhibitory pro-domain of MDC9 by a furin-type pro-protein convertase in the secretory pathway is a prerequisite for protease activity. After pro-domain removal, addnl. steps, such as protein kinase C-dependent phosphorylation, may be involved in regulating the catalytic activity of MDC9, which is likely to target different substrates than the related TNF-α-convertase.
REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:588912 CAPLUS
DOCUMENT NUMBER: 129:298070
TITLE: Synthetic matrix metalloproteinase inhibitors and tissue inhibitor of metalloproteinase (TIMP)-2, but not TIMP-1, inhibit shedding of tumor necrosis factor-α receptors in a human colon adenocarcinoma (Colo 205) cell line
AUTHOR(S): Lombard, Mark A.; Wallace, Tanya L.; Kubicek, Marc F.; Petzold, Gary L.; Mitchell, Mark A.; Hendges, Susan K.; Wilks, John W.

CORPORATE SOURCE: Cell and Molecular Biology, Pharmacia and Upjohn, Inc., Kalamazoo, MI, 49001, USA
SOURCE: Cancer Research (1998), 58(17), 4001-4007
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The solubilization of plasma membrane receptors through proteolytic cleavage of the ligand binding domain at the cell surface is an important mechanism for regulating cytokine function and receptor signaling. The inhibition of the shedding of a variety of receptors by synthetic inhibitors of the matrix metalloproteinases (MMPs) implicates metalloproteinases in this regulatory event. The authors examined the effects of two naturally occurring tissue inhibitors of metalloproteinases, TIMP-1 and TIMP-2, and several synthetic MMP inhibitors (MMPIs) on the shedding of both tumor necrosis factor α receptor type I (TNFα-RI; Mr 55,000) and TNFα-RII (Mr 75,000) by the Colo 205 human colon adenocarcinoma cell line. Culture of Colo 205 cells for 48 h resulted in the shedding of both TNFα-RI and TNFα-RII, as determined by ELISA. The shedding of TNFα receptors was not affected by TIMP-1 or protease inhibitors aprotinin, pepstatin, or leupeptin but was inhibited in a dose-dependent manner by the following synthetic MMPIs: batimastat and marimastat (BB-94 and BB-2516, resp., British Biotech, Inc.); CT1418 (Celltech Therapeutics); CGS27023A (Novartis Pharmaceuticals); and RO31-9790 (Roche), with IC50s ranging from 3.2 to 38.0 μM. Similarly, TIMP-2 from two different sources reproducibly inhibited the shedding of both TNFα-RI and TNFα-RII in a dose-dependent manner (IC50 = 286 nM for TNFα-RI shedding and 462 nM for shedding of TNFα-RII). The inhibition of TNFα-RI shedding was confirmed in the SW626 human ovarian adenocarcinoma cell line. The synthetic MMPIs and TIMP-2, but not TIMP-1, also caused a dose-dependent increase in the number of TNFα receptors retained on the surface of Colo 205 cells, as determined by flow cytometry. Inhibition of TNFα receptor shedding with TIMP-2 occurs at molar concns. 10-100 times less than those required with low mol. weight, synthetic MMPIs but at concns. greater than those required to inhibit collagen degradation Modulation of TNFα receptor shedding by TIMP-2 could have important implications for the pleiotropic effects of TNFα in both normal and malignant cells and for the pharmacol. activity of synthetic MMPIs.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:124446 CAPLUS
DOCUMENT NUMBER: 126:135633
TITLE: Arylsulfonamido-substituted hydroxamic acids for the treatment of tumors
INVENTOR(S): Macpherson, Lawrence Joseph; Parker, David Thomas
PATENT ASSIGNEE(S): Ciba-Geigy A.-G., Switz.
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9640101	A1	19961219	WO 1996-EP2418	19960604 <--
W: AL, AU, BB, BG, BR, CA, CN, CZ, EE, GE, HU, IL, IS, JP, KP, KR, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, TR,				

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            TT, UA, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR,
             IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML,
             MR, NE, SN, TD, TG
     US 5646167            A     19970708    US 1995-475166          19950607 <--
     AU 9661249            A     19961230    AU 1996-61249           19960604 <--
PRIORITY APPLN. INFO.:                       US 1995-475166       A  19950607
                                             US 1993-1136         A2 19930106
                                             US 1994-265296       A2 19940624
                                             US 1994-333676       A2 19941103
                                             WO 1996-EP2418       W  19960604
OTHER SOURCE(S):         MARPAT 126:135633
AB   The invention relates to the use of compds. NH(OH)COCR1R2N(CH2R)SO2X (X =
     carbocyclic or heterocyclic aryl; R, R1 = H, substituted lower alkyl,
     arylalkyl, biaryl, etc; R2 = H, lower alkyl) for the treatment of a
     tumor selected from human breast carcinoma, lung carcinoma,
     bladder carcinoma, colon carcinoma, prostate carcinoma, skin carcinoma,
     and ovarian carcinoma.  N-hydroxy-2(R)-[[4-methoxybenzenesulfonyl](3-
     picolyl)-amino]-3-methylbutanamide·HCl was prepared and formulated
     into a capsule.


=> l6 and (heat shock)
L6 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l6 and heat shock
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         59554 HEATS
       1427928 HEAT
                 (HEAT OR HEATS)
        153681 SHOCK
         11165 SHOCKS
        158688 SHOCK
                 (SHOCK OR SHOCKS)
         37818 HEAT SHOCK
                 (HEAT(W)SHOCK)
L7           2 L6 AND HEAT SHOCK

=> d l7 ibib abs

L7   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2004:166330  CAPLUS
DOCUMENT NUMBER:         141:17052
TITLE:                   Screening of stress enhancer based on analysis of gene
                         expression profiles: Enhancement of
                         hyperthermia-induced tumor necrosis by an
                         MMP-3 inhibitor
AUTHOR(S):               Kato, Naoki; Kobayashi, Takeshi; Honda, Hiroyuki
CORPORATE SOURCE:        Department of Biotechnology, School of Engineering,
                         Nagoya University, Nagoya, 464-8603, Japan
SOURCE:                  Cancer Science (2003), 94(7), 644-649
                         CODEN: CSACCM; ISSN: 1347-9032
PUBLISHER:               Japanese Cancer Association
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   To improve the therapeutic benefit of hyperthermia, we examined changes of
     global gene expression after heat shock using DNA
     microarrays consisting of 12,814 clones.  HeLa cells were treated for 1 h
     at 44° and RNA was extracted from the cells 0, 3, 6, and 12 h after
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heat shock. The 664 genes that were up or down-regulated after heat shock were classified into 7 clusters using fuzzy adaptive resonance theory (fuzzy ART). There were 41 genes in two clusters that were induced in the early phase after heat shock. In addition to shock response genes, such as hsp70 and hsp40, the stress response genes c-jun, c-fos and egr-1 were expressed in the early phase after heat shock. We also found that expression of matrix metalloproteinase 3 (MMP-3) was enhanced during the early response. We therefore investigated the role of MMP-3 in the heat shock response by examining HeLa cell survival after heat treatment in the presence and absence of an MMP-3 inhibitor, N-isobutyl-N-(4-methoxyphenyl-sulfonyl)glycylhydroxamic acid (NNGH) or N-hydroxy-2(R)-[[4-methoxysulfonyl](3-picolyl)amino]-3-methylbutaneamidehydrochloride(MMI270). The number of surviving cells 3 days after heat treatment significantly decreased, reaching 3.5% for NNGH and 0.2% for MMI270. These results indicate that the MMP-3 inhibitors enhanced heat shock-induced cell death and behaved as stress enhancers in cancer cells. This valuable conclusion was reached as a direct result of the gene expression profiling that was performed in these studies.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l7 ibib abs 1-2

L7 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:166330 CAPLUS

DOCUMENT NUMBER: 141:17052

TITLE: Screening of stress enhancer based on analysis of gene expression profiles: Enhancement of hyperthermia-induced tumor necrosis by an MMP-3 inhibitor

AUTHOR(S): Kato, Naoki; Kobayashi, Takeshi; Honda, Hiroyuki

CORPORATE SOURCE: Department of Biotechnology, School of Engineering, Nagoya University, Nagoya, 464-8603, Japan

SOURCE: Cancer Science (2003), 94(7), 644-649 CODEN: CSACCM; ISSN: 1347-9032

PUBLISHER: Japanese Cancer Association

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To improve the therapeutic benefit of hyperthermia, we examined changes of global gene expression after heat shock using DNA microarrays consisting of 12,814 clones. HeLa cells were treated for 1 h at 44° and RNA was extracted from the cells 0, 3, 6, and 12 h after heat shock. The 664 genes that were up or down-regulated after heat shock were classified into 7 clusters using fuzzy adaptive resonance theory (fuzzy ART). There were 41 genes in two clusters that were induced in the early phase after heat shock. In addition to shock response genes, such as hsp70 and hsp40, the stress response genes c-jun, c-fos and egr-1 were expressed in the early phase after heat shock. We also found that expression of matrix metalloproteinase 3 (MMP-3) was enhanced during the early response. We therefore investigated the role of MMP-3 in the heat shock response by examining HeLa cell survival after heat treatment in the presence and absence of an MMP-3 inhibitor, N-isobutyl-N-(4-methoxyphenyl-sulfonyl)glycylhydroxamic acid (NNGH) or N-hydroxy-2(R)-[[4-methoxysulfonyl](3-picolyl)amino]-3-methylbutaneamidehydrochloride(MMI270). The number of surviving cells 3 days after heat treatment significantly decreased, reaching 3.5% for NNGH and 0.2% for MMI270. These results indicate that the MMP-3 inhibitors enhanced heat shock-induced cell death and behaved as

stress enhancers in cancer cells. This valuable conclusion was reached as a direct result of the gene expression profiling that was performed in these studies.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS
DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307 <--
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606

PRIORITY APPLN. INFO.: GB 2002-5537 A 20020308
GB 2002-29054 A 20021212
WO 2003-EP2365 W 20030307

OTHER SOURCE(S): MARPAT 139:240351

AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l6 and radiotherapy
30601 RADIOTHERAPY
33 RADIOTHERAPIES
30617 RADIOTHERAPY
(RADIOTHERAPY OR RADIOTHERAPIES)
L8 3 L6 AND RADIOTHERAPY

=> d l8 ibib abs 1-3

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS

DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307 <--
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606

PRIORITY APPLN. INFO.: GB 2002-5537 A 20020308
GB 2002-29054 A 20021212
WO 2003-EP2365 W 20030307

OTHER SOURCE(S): MARPAT 139:240351
AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:551500 CAPLUS
DOCUMENT NUMBER: 139:117431
TITLE: 4,5-Diamino-1,2,3,4-tetrahydro-3,6-pyridazinediones as CXC chemokine receptor antagonists for treatment of inflammatory disorders and cancer
INVENTOR(S): Taveras, Arthur G.; Dwyer, Michael; Chao, Jianping; Baldwin, John J.; Merritt, Robert J.; Li, Ge; Chao, Jianhua; Yu, Younong
PATENT ASSIGNEE(S): Schering Corporation, USA; Pharmacopeia, Inc.
SOURCE: PCT Int. Appl., 210 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003057676	A1	20030717	WO 2003-US299	20030103 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SC, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VC, VN, YU, ZA, ZM
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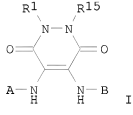
US 2004063709	A1	20040401	US 2003-335789	20030102	
US 6878709	B2	20050412			
CA 2472165	A1	20030717	CA 2003-2472165	20030103	<--
AU 2003207460	A1	20030724	AU 2003-207460	20030103	<--
EP 1461321	A1	20040929	EP 2003-705667	20030103	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
CN 1582280	A	20050216	CN 2003-801923	20030103	
JP 2005516029	T	20050602	JP 2003-557993	20030103	
MX 2004PA06555	A	20041004	MX 2004-PA6555	20040702	

PRIORITY APPLN. INFO.:

US 2002-346248P	P	20020104
US 2003-335789	A	20030102
WO 2003-US299	W	20030103

OTHER SOURCE(S): MARPAT 139:117431
GI



AB Prepns. for title compds. I [wherein R1 and R15 = independently H or (un)substituted (hetero)aryl, alkyl, (hetero)cycloalkyl(alkyl), or (hetero)arylalkyl; A = (un)substituted thiazolyl(alkyl), thienyl(alkyl), oxazolyl(alkyl), pyridinyl(alkyl), piperazinyl(alkyl), piperidinyl(alkyl), imidazolyl(alkyl), indolyl(alkyl), benzotriazolyl(alkyl), phenyl(alkyl), naphthyl(alkyl), carbamoylalkyl, etc.; B = (un)substituted Ph, benzotriazolyl, benzimidazolyl, indolyl, indazolyl, pyridinyl, pyrazolyl, thienyl, pyrrolyl, or pyrimidinyl; or pharmaceutically acceptable salts or solvates thereof] and their intermediates are disclosed (no data). In addition, CXCR1 SPA, CXCR2 SPA, calcium fluorescence, chemotaxis, and cytotoxicity assays are described. For example, 5-methylsalicylic acid was coupled with dimethylamine in the presence of DCC in EtOAc to give 2-hydroxy-N,N,5-trimethylbenzamide, which was nitrated (44%) and reduced using 10% Pd/C to give 3-amino-2-hydroxy-N,N,5-trimethylbenzamide (99%). The amine may be coupled with 1,2,3,4-tetrahydro-3,6-pyridazinediones to provide compds. of the invention (no data). I may exhibit a range of CXCR2 receptor binding activities from about 1 nM to about 10,100 nM. Thus, I and pharmaceutical compns. comprising I may be useful for the treatment of acute and chronic inflammatory disorders and cancer (no data).

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:359662 CAPLUS

DOCUMENT NUMBER: 137:306709
TITLE: Radiation-induced increase in invasive potential of human pancreatic cancer cells and its blockade by a matrix metalloproteinase inhibitor, CGS27023
AUTHOR(S): Qian, Li-Wu; Mizumoto, Kazuhiro; Urashima, Taro; Nagai, Eishi; Maehara, Naoki; Sato, Norihiro; Nakajima, Motowo; Tanaka, Masao
CORPORATE SOURCE: Department of Surgery and Oncology, Kyushu University, Fukuoka, 812-8582, Japan
SOURCE: Clinical Cancer Research (2002), 8(4), 1223-1227
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Purpose: Radiotherapy remains a major therapeutic option for patients with advanced pancreatic cancer. Nevertheless, the effects of irradiation on malignant biol. behaviors (e.g., migration and invasion of cancer cells) have yet to be clarified. Thus, we conducted an in vitro study to investigate the radiation-induced alterations around cell migration and invasion capacity. Experiment design: Three cell lines from human pancreatic cancer were included in the study. γ-Radiation was used for irradiation treatment. Cell migration and invasion ability were evaluated by Transwell migration assay and Matrigel invasion assay. The activity of MMP-2 and 9, and expression of urokinase-type plasminogen activator were investigated with gelatin zymog. and immunoblot, resp. Results: Irradiation enhances invasive potential in some pancreatic cancer cells, whereas it significantly inhibits cell proliferation and migration. This hitherto unknown biol. effect of irradiation involves enhanced matrix metalloproteinase (MMP)-2 activity. Consequently, simultaneous administration of an MMP inhibitor, CGS27023A, suppresses the radiation-enhanced invasion through blockade of transition of MMP-2 from latent type to active type. Conclusion: Because radiation may increase invasion ability through activating MMP proteolytic system, simultaneous administration of the MMP inhibitor during radiotherapy could be a potent adjuvant therapeutic approach to improve the efficacy of radiotherapy for pancreatic cancer.
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> s heat shock therapy
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         59554 HEATS
       1427928 HEAT
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        153681 SHOCK
         11165 SHOCKS
        158688 SHOCK
                 (SHOCK OR SHOCKS)
        342832 THERAPY
         32934 THERAPIES
        360689 THERAPY
                 (THERAPY OR THERAPIES)
L9            7 HEAT SHOCK THERAPY
                (HEAT(W)SHOCK(W)THERAPY)

=> s heat shock
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        158688 SHOCK
                 (SHOCK OR SHOCKS)
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                 (HEAT(W)SHOCK)

=> s l10 and (matrix metalloproteinase)
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        594341 MATRIX
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         11763 METALLOPROTEINASES
         29364 METALLOPROTEINASE
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         22325 MATRIX METALLOPROTEINASE
                 (MATRIX(W)METALLOPROTEINASE)
L11        324 L10 AND (MATRIX METALLOPROTEINASE)

=> d l9 ibib abs 1-7

L9   ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2008:192635  CAPLUS
TITLE:                    HSP72 protects against obesity-induced insulin
                          resistance
AUTHOR(S):                Chung, Jason; Nguyen, Anh-Knoi; Henstridge, Darren C.;
                          Holmes, Anna G.; Chan, M. H. Stanley; Mesa, Jose L.;
                          Lancaster, Graeme I.; Southgate, Robert J.; Bruce,
                          Clinton R.; Duffy, Stephen J.; Horvath, Ibolya;
                          Mestril, Ruben; Watt, Matthew J.; Hooper, Philip L.;
                          Kingwell, Bronwyn A.; Vigh, Laszlo; Hevener, Andrea;
                          Febbraio, Mark A.
CORPORATE SOURCE:         Cellular and Molecular Laboratory, baker Heart
                          Research Institute, Prahran, 8008, Australia
SOURCE:                   Proceedings of the National Academy of Sciences of the
                          United States of America (2008), 105(5), 1739-1744
                          CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER:                National Academy of Sciences
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Patients with type 2 diabetes have reduced gene expression of heat shock
     protein (HSP) 72, which correlates with reduced insulin sensitivity.  Heat
     therapy, which activates HSP72, improves clin. parameters in these
     patients.  Activation of several inflammatory signaling proteins such as
     c-jun amino terminal kinase (JNK), inhibitor of κB kinase, and tumor
     necrosis factor-α, can induce insulin resistance, but HSP 72 can
     block the induction of these mols. in vitro.  Accordingly, we examined
     whether activation of HSP72 can protect against the development of insulin
     resistance.  First, we show that obese, insulin resistant humans have
     reduced HSP72 protein expression and increased JNK phosphorylation in
     skeletal muscle.  We next used heat shock
     therapy, transgenic overexpression, and pharmacol. means to
     overexpress HSP72 either specifically in skeletal muscle or globally in
     mice.  Herein, we show that regardless of the means used to achieve an
     elevation in HSP72 protein, protection against diet- or obesity-induced
     hyperglycemia, hyperinsulinemia, glucose intolerance, and insulin
     resistance was observed  This protection was tightly associated with the
     prevention of JNK phosphorylation.  These findings identify an essential
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role for HSP72 in blocking inflammation and preventing insulin resistance in the context of genetic obesity or high-fat feeding.

L9 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:102747 CAPLUS
TITLE: HSP72 protects against obesity-induced insulin resistance
AUTHOR(S): Chung, Jason; Nguyen, Anh-Khoi; Henstridge, Darren C.; Holmes, Anna G.; Chan, M. H. Stanley; Mesa, Jose L.; Lancaster, Graeme I.; Southgate, Robert J.; Bruce, Clinton R.; Duffy, Stephen J.; Horvath, Ibolya; Mestril, Ruben; Watt, Matthew J.; Hooper, Philip L.; Kingwell, Bronwyn A.; Vigh, Laszlo; Hevener, Andrea; Febbraio, Mark A.
CORPORATE SOURCE: Cellular and Molecular Metabolism Lab., Baker Heart Research Institute, Prahran, Victoria, 8008, Australia
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, Early Edition (2008), (Jan 25 2008), 1-6, 6 pp.
CODEN: PNASC8
URL: http://www.pnas.org/cgi/reprint/0705799105v1
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Patients with type 2 diabetes have reduced gene expression of heat shock protein (HSP) 72, which correlates with reduced insulin sensitivity. Heat therapy, which activates HSP72, improves clin. parameters in these patients. Activation of several inflammatory signaling proteins such as c-jun amino terminal kinase (JNK), inhibitor of κB kinase, and tumor necrosis factor-, can induce insulin resistance, but HSP 72 can block the induction of these mols. in vitro. Accordingly, we examined whether activation of HSP72 can protect against the development of insulin resistance. First, we show that obese, insulin resistant humans have reduced HSP72 protein expression and increased JNK phosphorylation in skeletal muscle. We next used heat shock therapy, transgenic overexpression, and pharmacol. means to overexpress HSP72 either specifically in skeletal muscle or globally in mice. Herein, we show that regardless of the means used to achieve an elevation in HSP72 protein, protection against diet- or obesity-induced hyperglycemia, hyperinsulinemia, glucose intolerance, and insulin resistance was observed This protection was tightly associated with the prevention of JNK phosphorylation. These findings identify an essential role for HSP72 in blocking inflammation and preventing insulin resistance in the context of genetic obesity or high-fat feeding.

L9 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:60754 CAPLUS
Correction of: 2004:1036571
DOCUMENT NUMBER: 142:233342
Correction of: 142:16836
TITLE: Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 33
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2007031841	A1	20070208	US 2003-601518	20030620
US 2006134635	A1	20060622	US 2004-802875	20040312
US 2005191637	A1	20050901	US 2004-803737	20040318
US 2005196762	A1	20050908	US 2004-803759	20040318
US 2005196763	A1	20050908	US 2004-803857	20040318
US 2005196764	A1	20050908	US 2004-803858	20040318
US 2005208505	A1	20050922	US 2004-803648	20040318
US 2005208519	A1	20050922	US 2004-989191	20041115
PRIORITY APPLN. INFO.:			US 1999-115125P	P 19990106
			US 2000-477148	B1 20000104
			US 2002-268730	A2 20021009
			US 2003-601518	A2 20030620
			US 2004-802875	A2 20040312
			US 2001-271955P	P 20010228
			US 2001-275017P	P 20010312
			US 2001-305340P	P 20010713
			US 2002-85783	A2 20020228
			US 2004-812731	A2 20040330
			WO 2004-US20836	A2 20040621

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L9 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:770029 CAPLUS
DOCUMENT NUMBER: 141:258763
TITLE: Genes showing altered patterns of expression in metastatic lung and breast cancer and their use in diagnosis and therapy
INVENTOR(S): Aziz, Natasha; Zlotnik, Albert
PATENT ASSIGNEE(S): Protein Design Labs, Inc., USA
SOURCE: PCT Int. Appl., 233 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004063355	A2	20040729	WO 2004-XA885	20040112

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,

BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
WO 2004063355 A2 20040729 WO 2004-US885 20040112
WO 2004063355 A3 20050929
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2003-439058P P 20030110
WO 2004-US885 A 20040112

AB Genes that showed altered patterns of expression in metastatic lung and breast cancer are described for use in diagnosis and prognosis of the diseases. The genes or gene products may also be useful as targets for anti-cancer drugs. [This abstract record is one of 4 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L9 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:39697 CAPLUS
DOCUMENT NUMBER: 140:123703
TITLE: Human prostate cancer marker genes associated with various metastatic stages identified by gene profiling, and related compositions, kits, and methods for diagnosis, prognosis and therapy
INVENTOR(S): Schlegel, Robert; Endege, Wilson O.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 131 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004009481	A1	20040115	US 2002-166883	20020611
US 2004009481	A1	20040115	US 2002-166883	20020611
US 2004009481	A1	20040115	US 2002-166883	20020611
US 2004009481	A1	20040115	US 2002-166883	20020611
US 2004009481	A1	20040115	US 2002-166883	20020611

PRIORITY APPLN. INFO.: US 2001-297285P P 20010611
US 2002-166883 A 20020611

AB The invention relates to compns., kits, and methods for diagnosing, staging, prognosing, monitoring and treating human prostate cancers. A variety of marker genes are provided, wherein changes in the levels of expression of one or more of the marker genes is correlated with the presence of prostate cancer. In particular, three sets of the marker genes set, corresponding to 11617 GenBank Accession Nos. (only 2168 new submissions) and 15 SEQ IDs, are identified by transcription profiling using RNA derived from clin. samples, that were expressed at least 2-fold or greater than the normal controls. Using TNM staging approach, these markers are divided to three groups, ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the liver (M stage); ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the bone (M stage); and ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the lymph nodes (N stage and/or M stage). The

invention also relates to a kit for assessing the specific type of metastatic prostate cancer, e.g., cancer that has metastasized to the liver, bone or lymph nodes. [This abstract record is one of three records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L9 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:348346 CAPLUS
TITLE: Heat shock does not attenuate low-frequency fatigue
AUTHOR(S): Thomas, J. A.; Noble, E. G.
CORPORATE SOURCE: School of Kinesiology, The University of Western Ontario, London, ON, N6A 3K7, Can.
SOURCE: Canadian Journal of Physiology and Pharmacology (1999), 77(1), 64-70
CODEN: CJPPA3; ISSN: 0008-4212
PUBLISHER: National Research Council of Canada
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Whole-body hyperthermia or heat shock confers protection to myocardial contractility against reperfusion-induced injury. The purpose of this study was to determine whether heat shock could provide similar protection to skeletal muscle contractility against low-frequency fatigue. Male Sprague-Dawley rats (6 rats/group) were heat shocked at 41.5°C for 15 min either 24 h or 4 days prior to fatiguing stimulation to compare the contractile responses of the plantaris muscle with those of a nonheated group. Both 24 h and 4 days after heat shock, the 72-kDa heat shock protein (HSP72) was elevated above control levels. There were no differences between the heat-shocked and non-heat-shocked animals in measures of contractility prior to fatiguing contractions or in resistance to fatigue. Heat-shock preconditioning did not lead to improved postfatigue force recovery above control responses and, in fact, delayed the recovery of force. This study does not support the use of heat-shock therapy to improve skeletal muscle contractile performance under fatiguing conditions.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1993:406516 CAPLUS
DOCUMENT NUMBER: 119:6516
TITLE: Alterations in nuclear protein mass and macromolecular synthesis following heat shock
AUTHOR(S): Higashikubo, Ryuji; Roti Roti, Joseph L.
CORPORATE SOURCE: Sch. Med., Washington Univ., St. Louis, MO, 63108, USA
SOURCE: Radiation Research (1993), 134(2), 193-201
CODEN: RAREAE; ISSN: 0033-7587
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Possible correlations between post-heat alterations in nuclear protein mass and the resumption of macromol. (DNA, RNA, and protein) synthesis were investigated in CHO cells. Nuclear protein content was measured using flow cytometry. Macromol. synthesis was measured by incorporation of radioactively labeled precursors into TCA-precipitable material of whole cells and isolated nuclei. Following an initial increase which was dependent on the heat dose, nuclear protein mass decreased following a monotonic function which appeared to be multiphasic. The synthesis of DNA, RNA, and protein was inhibited in a manner dependent on the heat dose and remained suppressed for an interval dependent on the heat dose and remained suppressed for an interval dependent on the heat dose before recovery. The kinetics of resumption of DNA, RNA, and protein synthesis correlated linearly with the nuclear protein mass measured immediately after heating. Also, the time of onset of recovery of RNA and protein

syntheses correlated linearly with the time at which nuclear protein mass returned to 125% of control, a level which has been implicated as a possible threshold in previous studies. More significantly, the onset of the resumption of DNA synthesis showed a one-to-one correlation with the time at which the nuclear protein mass returned to 125% of control. These correlations suggest that there may be causal relationships between the resumption of DNA, RNA, and protein synthesis and the reduction of the amount of nuclear protein binding, particularly in the case of DNA synthesis.

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The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

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766514 RADIATION
13479 RADIATIONS
772062 RADIATION
(RADIATION OR RADIATIONS)
869 CHEMORADIOTHERAPY
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=> d l12

L12 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2003:737608 CAPLUS
DN 139:240351
TI Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
IN Nakajima, Motowo
PA Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SO PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003075959	A1	20030918	WO 2003-EP2365	20030307 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
	RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
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	EP 1485131	A1	20041215	EP 2003-709764	20030307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2005526760	T	20050908	JP 2003-574232	20030307
	US 2005232915	A1	20051020	US 2005-506936	20050606
PRAI	GB 2002-5537	A	20020308		
	GB 2002-29054	A	20021212		
	WO 2003-EP2365	W	20030307		

OS MARPAT 139:240351
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l12 ibib abs 1-3

L12 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS
DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307 <--
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606

PRIORITY APPLN. INFO.:
GB 2002-5537 A 20020308
GB 2002-29054 A 20021212
WO 2003-EP2365 W 20030307

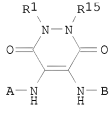
OTHER SOURCE(S): MARPAT 139:240351
AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:551500 CAPLUS
DOCUMENT NUMBER: 139:117431
TITLE: 4,5-Diamino-1,2,3,4-tetrahydro-3,6-pyridazinediones as CXC chemokine receptor antagonists for treatment of inflammatory disorders and cancer
INVENTOR(S): Taveras, Arthur G.; Dwyer, Michael; Chao, Jianping; Baldwin, John J.; Merritt, Robert J.; Li, Ge; Chao, Jianhua; Yu, Younong
PATENT ASSIGNEE(S): Schering Corporation, USA; Pharmacopeia, Inc.
SOURCE: PCT Int. Appl., 210 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003057676	A1	20030717	WO 2003-US299	20030103 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LU, LV, MA, MD, MG, MK, MN, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SC, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UZ, VC, VN, YU, ZA, ZM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 20040063709	A1	20040401	US 2003-335789	20030102
US 6878709	B2	20050412		
CA 2472165	A1	20030717	CA 2003-2472165	20030103 <--
AU 2003207460	A1	20030724	AU 2003-207460	20030103 <--
EP 1461321	A1	20040929	EP 2003-705667	20030103
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1582280	A	20050216	CN 2003-801923	20030103
JP 2005516029	T	20050602	JP 2003-557993	20030103
MX 2004PA06555	A	20041004	MX 2004-PA6555	20040702
PRIORITY APPLN. INFO.:			US 2002-346248P	P 20020104
			US 2003-335789	A 20030102
			WO 2003-US299	W 20030103

OTHER SOURCE(S): MARPAT 139:117431
GI



I

AB Prepns. for title compds. I [wherein R1 and R15 = independently H or (un)substituted (hetero)aryl, alkyl, (hetero)cycloalkyl(alkyl), or (hetero)arylalkyl; A = (un)substituted thiazolyl(alkyl), thienyl(alkyl), oxazolyl(alkyl), pyridinyl(alkyl), piperazinyl(alkyl), piperidinyl(alkyl), imidazolyl(alkyl), indolyl(alkyl), benzotriazolyl(alkyl), phenyl(alkyl), naphthyl(alkyl), carbamoylalkyl, etc.; B = (un)substituted Ph, benzotriazolyl, benzimidazolyl, indolyl, indazolyl, pyridinyl, pyrazolyl, thienyl, pyrrolyl, or pyrimidinyl; or pharmaceutically acceptable salts or solvates thereof] and their intermediates are disclosed (no data). In addition, CXCR1 SPA, CXCR2 SPA, calcium fluorescence, chemotaxis, and cytotoxicity assays are described. For example, 5-methylsalicylic acid was coupled with dimethylamine in the presence of DCC in EtOAc to give 2-hydroxy-N,N,5-trimethylbenzamide, which was nitrated (44%) and reduced using 10% Pd/C to give 3-amino-2-hydroxy-N,N,5-trimethylbenzamide (99%). The amine may be coupled with 1,2,3,4-tetrahydro-3,6-pyridazinediones to provide compds. of the invention (no data). I may exhibit a range of

CXCR2 receptor binding activities from about 1 nM to about 10,100 nM. Thus, I and pharmaceutical compns. comprising I may be useful for the treatment of acute and chronic inflammatory disorders and cancer (no data).

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:359662 CAPLUS

DOCUMENT NUMBER: 137:306709

TITLE: Radiation-induced increase in invasive potential of human pancreatic cancer cells and its blockade by a matrix metalloproteinase inhibitor, CGS27023

AUTHOR(S): Qian, Li-Wu; Mizumoto, Kazuhiro; Urashima, Taro; Nagai, Eishi; Maehara, Naoki; Sato, Norihiro; Nakajima, Motowo; Tanaka, Masao

CORPORATE SOURCE: Department of Surgery and Oncology, Kyushu University, Fukuoka, 812-8582, Japan

SOURCE: Clinical Cancer Research (2002), 8(4), 1223-1227
CODEN: CCREF4; ISSN: 1078-0432

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Purpose: Radiotherapy remains a major therapeutic option for patients with advanced pancreatic cancer. Nevertheless, the effects of irradiation on malignant biol. behaviors (e.g., migration and invasion of cancer cells) have yet to be clarified. Thus, we conducted an in vitro study to investigate the radiation-induced alterations around cell migration and invasion capacity. Experiment design: Three cell lines from human pancreatic cancer were included in the study. γ- Radiation was used for irradiation treatment. Cell migration and invasion ability were evaluated by Transwell migration assay and Matrigel invasion assay. The activity of MMP-2 and 9, and expression of urokinase-type plasminogen activator were investigated with gelatin zymog. and immunoblot, resp. Results: Irradiation enhances invasive potential in some pancreatic cancer cells, whereas it significantly inhibits cell proliferation and migration. This hitherto unknown biol. effect of irradiation involves enhanced matrix metalloproteinase (MMP)-2 activity. Consequently, simultaneous administration of an MMP inhibitor, CGS27023A, suppresses the radiation-enhanced invasion through blockade of transition of MMP-2 from latent type to active type. Conclusion: Because radiation may increase invasion ability through activating MMP proteolytic system, simultaneous administration of the MMP inhibitor during radiotherapy could be a potent adjuvant therapeutic approach to improve the efficacy of radiotherapy for pancreatic cancer.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s metalloproteinase and heat shock

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        26797 METALLOPROTEINASE
        11763 METALLOPROTEINASES
        29364 METALLOPROTEINASE
              (METALLOPROTEINASE OR METALLOPROTEINASES)
      1410582 HEAT
        59554 HEATS
      1427928 HEAT
              (HEAT OR HEATS)
       153681 SHOCK
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        11165 SHOCKS
       158688 SHOCK
                (SHOCK OR SHOCKS)
        37818 HEAT SHOCK
                (HEAT(W)SHOCK)
L13       443 METALLOPROTEINASE AND HEAT SHOCK

=> s l13 and radiother? or radiation or chemoradiotherapy
        31430 RADIOTHER?
       766514 RADIATION
        13479 RADIATIONS
       772062 RADIATION
                (RADIATION OR RADIATIONS)
          869 CHEMORADIOTHERAPY
L14    772590 L13 AND RADIOTHER? OR RADIATION OR CHEMORADIOTHERAPY

=> s l13 and (radiother? or radiation or chemoradiotherapy)
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       766514 RADIATION
        13479 RADIATIONS
       772062 RADIATION
                (RADIATION OR RADIATIONS)
          869 CHEMORADIOTHERAPY
L15        18 L13 AND (RADIOTHER? OR RADIATION OR CHEMORADIOTHERAPY)

=> d l15 ibib abs 1-15

L15  ANSWER 1 OF 18  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2007:433685  CAPLUS
DOCUMENT NUMBER:          146:460567
TITLE:                    Nucleic acid vaccines encoding matrix
                          metalloproteinase 11 and immunoenhancing
                          element against cancer or carcinoma
INVENTOR(S):              Aurisicchio, Luigi; La Monica, Nicola
PATENT ASSIGNEE(S):       Istituto di Ricerche di Biologia Molecolare P.
                          Angeletti S.p.A., Italy; Ciliberto, Gennaro; Lazzaro,
                          Domenico; Mori, Federica; Peruzzi, Daniela
SOURCE:                   PCT Int. Appl., 68pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2007042169        A2    20070419    WO 2006-EP9536         20061003
     WO 2007042169        A3    20070531
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
             KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
             MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS,
             RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRIORITY APPLN. INFO.:                       US 2005-724498P     P  20051007
AB   Compns. comprising matrix metalloproteinase 11 (MMP-11) or
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stromelysin-3 (ST-3) or the nucleic acid encoding the MMP-11 for use in vaccines for treating tumors and cancers, which overexpress MMP-11, are described. In particular embodiments, the compns. comprise a nucleic acid encoding a fusion polypeptide that includes the catalytically inactivated MMP-11 linked at the C-terminus to an immunoenhancing element wherein the codons encoding the MMP-11 and the immunoenhancing element have been optimized for enhanced expression of the fusion polypeptide in human cells. In other embodiments, the compns. comprise the catalytically inactivated MMP-11 linked at the C-terminus to an immunoenhancing element. The compns. can be used alone or in synergy with vaccines against other tumor associated antigens as well as with conventional therapies such as radiation therapy and chemotherapy.

L15 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:1157631 CAPLUS
DOCUMENT NUMBER: 145:483673
TITLE: Novel methods and devices for evaluating poisons
INVENTOR(S): Ching, Edwin P.; Johnson, Dale E.; Sudarsanam, Sucha
PATENT ASSIGNEE(S): Emiliem, USA
SOURCE: PCT Int. Appl., 132pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006116622	A2	20061102	WO 2006-US16067	20060426
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006253262	A1	20061109	US 2006-380388	20060426
EP 1880332	A2	20080123	EP 2006-751675	20060426
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
PRIORITY APPLN. INFO.:			US 2005-675741P	P 20050427
			US 2006-778133P	P 20060301
			WO 2006-US16067	W 20060426

AB Methods and devices useful for evaluating poisons or other chemical entities, and for using such methods to forecast unfavorable drug effects. The present invention provides lists of biomarkers for anal., either directly or indirectly, which affect the toxicity pathways. These may be evaluated at many levels, including genetic, genotyping, evaluation of combination pairing of diploid alleles or haplotypes, RNA expression, protein expression, functional activity, posttranslational anal. or evaluation, etc. Thus, the biomarkers refer to the corresponding genetic information, RNA, protein, or other structural embodiments thereof. And the means to use these biomarkers, e.g., to evaluate status of toxicity pathways, to evaluate individual risk or susceptibility to various toxic pathways from exposure or therapeutic intervention, to generate test systems for drug development, are all provided by identifying critical and significant

contributors to the pathway progression. The present invention is directed to accelerating the speed of development and reducing the resource investment necessary to determine these features for directing use of such substances or treatments to appropriate biol. contexts.

L15 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:816927 CAPLUS
DOCUMENT NUMBER: 145:267789
TITLE: The dynamic phase of cancer cells by the low temperature narrow wavelength far infrared radiation
AUTHOR(S): Hosokawa, Hiroyoshi
CORPORATE SOURCE: Dep. Oral Maxill. Surg., Grad. Sch. Dentistry, the University of Tokuyama, Japan
SOURCE: Shikoku Shigakkai Zasshi (2006), 19(1), 35-54
CODEN: SSZAED; ISSN: 0914-6091
PUBLISHER: Shikoku Shigakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Far IR ray (FIR) are known to have some effects on the human body, but little is known about the non-fever effects in normal thermal fields. We developed a CO2 incubator and an animal raiser that is able to radiate low temperature narrow wavelength (limited) FIR at wavelength of 4 to 20 μm with a peak wavelength 7 to 12 μm, which had strong effects on living tissue, and we investigated the effects of this FIR on cancer cells. In in vitro analyses, analyses of cell proliferation and cell cycle were carried out using 5-bromo-2'-deoxy-uridine (BrdU) incorporation and flow cytometry in three cancer cell lines: the human vulval epidermal cell line A431, the human tongue squamous cell carcinoma (SCC) line HSC3, and the human gingiva SCC line Sa3. In addition, from the viewpoint of the heat shock proteins (HSPs), especially the HSP70 protein, having cytoprotection for various stresses, Hsp70A gene expression was examined using real-time reverse transcription polymerase chain reaction. The effect of HSP70 protein on cell proliferation for limited FIR was analyzed by transfecting Hsp70A expression vector or by repressing Hsp70A and Hsp70C mRNA using gene silencing methods with siRNA. In in vivo analyses, we generated xenograft tumors of A431 and Sa3 cells and examined the changes of tumor volume, genetic alteration and histol. observation. As a result, limited FIR suppressed cell proliferation of HSC3 and Sa3 cells, not A431 cells. The cell cycle of HSC3 cells was mainly delayed by limited FIR in the G2/M stage, while necrotic cells of Sa3 cells slightly increased by limited FIR. Moreover, the expression of Hsp70A gene and HSP70 protein was higher on A431 cells whose cell proliferation was not suppressed by limited FIR. On BrdU incorporation anal. under the condition in which HSP70 protein was repressed, BrdU incorporation of A431 cells was suppressed. In in vivo analyses, limited FIR suppressed both the growth of A431 tumor and Sa3 tumor. Tumor tissues of A431 in limited FIR group were encapsulated and matrix metalloproteinase (MMP)-1, -9, -10, -13 were significantly suppressed in the protein level. On the other hand, limited FIR induced the apoptosis in the Sa3 tumor. These findings in vitro suggest that limited FIR suppressed the proliferation of certain cancer cells, and the suppressive effect depended on expression level of HSP70 protein. These findings in vivo that limited FIR suppressed the tumor growth of A431 by inhibiting MMPs, and that of Sa3 by inducing apoptosis.

L15 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:167588 CAPLUS
DOCUMENT NUMBER: 144:254148
TITLE: Aminopteridinones as anticancer agents, their preparation, pharmaceutical compositions, and use in therapy

INVENTOR(S): Munzert, Gerd; Steegmaier, Martin; Baum, Anke
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE: PCT Int. Appl., 158 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006018182	A1	20060223	WO 2005-EP8623	20050809
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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US 2006058311	A1	20060316	US 2005-189540	20050726
AU 2005274384	A1	20060223	AU 2005-274384	20050809
CA 2576269	A1	20060223	CA 2005-2576269	20050809
EP 1827441	A1	20070905	EP 2005-770228	20050809
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BA, HR, YU				
CN 101039673	A	20070919	CN 2005-80035272	20050809
IN 2007DN00888	A	20070803	IN 2007-DN888	20070202
MX 200701853	A	20070328	MX 2007-1853	20070214
KR 2007050478	A	20070515	KR 2007-705955	20070314

PRIORITY APPLN. INFO.:
EP 2004-19361 A 20040814
EP 2004-19448 A 20040817
WO 2005-EP8623 W 20050809

OTHER SOURCE(S): MARPAT 144:254148
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The invention relates to a group of aminopteridinones I, which are useful for the treatment of diseases which involve cell proliferation. In compds. I, R1 and R2 are independently selected from H and (un)substituted C1-6 alkyl, or R1 and R2 together form a 2- to 5-membered alkylene bridge, optionally containing 1 or 2 heteroatoms; R3 is (un)substituted C1-12 alkyl, C2-12 alkenyl, C2-12 alkynyl, C6-14 aryl, etc.; R4 is H, OH, CN, halo, (un)substituted amino, (un)substituted C1-6 alkyl, C1-5 alkoxy, etc.; L is (un)substituted C2-10 alkylene, (un)substituted C2-10 alkenylene, (un)substituted C6-14 arylene, etc.; R5 is (un)substituted morpholinyl, (un)substituted piperidinyl, (un)substituted piperazinyl, (un)substituted piperazinylcarbonyl, (un)substituted pyrrolidinyl, (un)substituted thiomorpholinyl, etc.; n is 0 or 1; and m is 1 or 2; including tautomers, stereoisomers, salts, solvates, polymorphs, and prodrugs thereof. The invention also relates to the preparation of I, pharmaceutical compns. comprising a compound I, at least one other therapeutic agent, optionally

with one or more pharmaceutically acceptable excipients, as well as to the use of the compns. for the treatment of diseases which involve cell proliferation, migration or apoptosis of cancer cells, or angiogenesis. Esterification of (R)-2-aminobutyric acid and reductive condensation with cyclopentanone gave cyclopentylamine II, which underwent regioselective substitution of 2,4-dichloro-5-nitropyrimidine and reductive heterocyclization to form pteridinone III. N-Methylation of III followed by substitution with 4-amino-3-methoxybenzoic acid and amidation with 1-methyl-4-aminopiperidine resulted in the formation of aminopteridinone IV. A combination of suboptimal doses of irinotecan and compound IV shows an additive/synergistic effect in a human colon carcinoma model and is well tolerated. Meanwhile, compound IV acts at least additively with docetaxel in a human non-small cell lung carcinoma model and not antagonistically with gemcitabine in a human adenocarcinoma model.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:151685 CAPLUS
DOCUMENT NUMBER: 144:309813
TITLE: The receptor for advanced glycation end products is highly expressed in the skin and upregulated by advanced glycation end products and tumor necrosis factor-alpha
AUTHOR(S): Lohwasser, Christina; Neureiter, Daniel; Weigle, Bernd; Kirchner, Thomas; Schuppan, Detlef
CORPORATE SOURCE: Department of Medicine I, University of Erlangen-Nuernberg, Germany
SOURCE: Journal of Investigative Dermatology (2006), 126(2), 291-299
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Advanced glycation end products (AGEs) form non-enzymically from reactions of proteins with reducing sugars. In the skin, AGEs were reported to accumulate in dermal elastin and collagens and to interact nonspecifically with the cell membrane of dermal fibroblasts. Therefore, AGEs may influence the process of skin aging. We investigated the presence of the AGE receptor RAGE in skin and the influence of AGEs on receptor expression and the formation of extracellular matrix (ECM). Sections of sun-protected and sun-exposed skin were analyzed with monoclonal antibodies against (RAGE), heat-shock protein 47, factor XIIIa, CD31, and CD45. RAGE was mainly expressed in fibroblasts, dendrocytes, and keratinocytes and to a minor extent in endothelial and mononuclear cells. Human foreskin fibroblasts (HFFs) highly expressed RAGE on the protein and mRNA level when analyzed by quant. Western blotting and real-time PCR. Incubation of HFFs with the specific RAGE ligand Nε-(carboxymethyl)lysine-modified BSA (CML-BSA) and tumor necrosis factor-alpha resulted in significant upregulation of RAGE expression. CML-BSA induced a mildly profibrogenic pattern, increasing connective tissue growth factor, transforming growth factor-beta (TGF-β) 1, and procollagen-α1(I) mRNA, whereas expression of matrix metalloproteinase (MMP)-1, -2, -3, and -12 was unaffected. We conclude that in HFFs, AGE-RAGE interactions may influence the process of skin aging through mild stimulation of ECM gene expression.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:120414 CAPLUS
DOCUMENT NUMBER: 144:184702

TITLE: Gene expression profiles for identifying patients at risk of developing encephalitis following immunotherapy for Alzheimer's disease

INVENTOR(S): O'Toole, Margot; Dorner, Andrew J.; Janszen, Derek B.; Slonim, Donna K.; Mounts, William M.; Reddy, Padmalatha S.; Hill, Andrew A.

PATENT ASSIGNEE(S): Wyeth, USA

SOURCE: PCT Int. Appl., 298 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006014755	A2	20060209	WO 2005-US25771	20050720
WO 2006014755	A3	20060413		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2571856	A1	20060209	CA 2005-2571856	20050720
US 2006073496	A1	20060406	US 2005-186236	20050720
EP 1784509	A2	20070516	EP 2005-795582	20050720
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

PRIORITY APPLN. INFO.:

US 2004-589877P	P	20040720
US 2005-672716P	P	20050418
WO 2005-US25771	W	20050720

AB The present invention generally relates to a method for an improved treatment for Alzheimer's disease (AD) using immunotherapy, e.g., immunotherapy targeting β amyloid (Aβ) and immunotherapy based on AN1792. By ANOVA and GeneCluster analyses of Affymetrix U133A GeneChip data, statistically significant assocns. were detected between the gene expression profiles of peripheral blood mononuclear cells of patients prior to immunization with AN1792 and the post-immunization development of encephalitis. In addition, statistically significant assocns. were found between the pre-immunization gene expression profile in PBMCs and post-immunization development of IgG response. The method allows for predicting an adverse clin. response, and therefore allows for an improved safety profile of AN1792. In another embodiment, the method allows for predicting a favorable clin. response, and therefore allows for an improved efficacy profile of AN1792. The methods of the present invention may be combined to predict a favorable clin. response and the lack of an adverse clin. response.

L15 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2005:1293778 CAPLUS

DOCUMENT NUMBER: 144:35066

TITLE: Gene expression profiling in the prostate in the diagnosis and Gleason staging of high- and low-grade tumors

INVENTOR(S): Shekar, Mamatha; Zhang, Zhaomei; Caldwell, Mitchell

C.; Chen, Zuxiong; Fan, Zhenbin; McNeal, John E.; Nolley, Rosalie; Stamey, Thomas A.; Warrington, Janet A.; Palma, John F.
PATENT ASSIGNEE(S): Affymetrix, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 411,537.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2005272052	A1	20051208	US 2004-975592		20041027
US 2004029151	A1	20040212	US 2003-411537		20030409
PRIORITY APPLN. INFO.:			US 2002-371304P	P	20020409
			US 2003-411537	A2	20030409

AB Many genes are affected in prostate cancers which have not been previously identified. This includes genes that have been up-regulated or down-regulated. Monitoring the expression levels of these genes is useful to identify the existence of prostate cancer and to differentiate low-risk (Gleason grade 3), and high risk (Gleason grade 4 or 5) tumors. Also, monitoring the expression levels of these genes is useful to predict the effectiveness of treatment, outcome, use of therapeutics, and screening drugs useful for the treatment of prostate cancer.

L15 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:493568 CAPLUS
DOCUMENT NUMBER: 143:48169
TITLE: Implantable sensors and pumps and anti-scarring agents
INVENTOR(S): Hunter, William L.; Gravett, David M.; Toleikis, Philip M.; Maiti, Arpita
PATENT ASSIGNEE(S): Angiotech International A.-G., Switz.
SOURCE: PCT Int. Appl., 1619 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 19
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005051871	A2	20050609	WO 2004-US39387	20041122
WO 2005051871	A9	20060727		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005148512	A1	20050707	US 2004-986230	20041110
US 2005181977	A1	20050818	US 2004-986231	20041110
CN 101094613	A	20071226	CN 2004-80031664	20041110
AU 2004293463	A1	20050609	AU 2004-293463	20041122
CA 2536242	A1	20050609	CA 2004-2536242	20041122
WO 2005051232	A2	20050609	WO 2004-US39346	20041122

WO 2005051232 A3 20051208
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2006055008 A2 20060526 WO 2004-US39353 20041122
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

EP 1685085 A2 20060802 EP 2004-817879 20041122
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS

CN	1878514	A	20061213	CN 2004-80033341	20041122
JP	2007513650	T	20070531	JP 2006-541669	20041122
US	2005149158	A1	20050707	US 2004-409	20041129
US	2005175662	A1	20050811	US 2004-451	20041129
US	2005175661	A1	20050811	US 2004-999205	20041129
US	2005186243	A1	20050825	US 2004-97	20041129
US	2005186242	A1	20050825	US 2004-999204	20041129
US	2005191331	A1	20050901	US 2004-1419	20041130
US	2005175663	A1	20050811	US 2004-1791	20041202
US	2005181008	A1	20050818	US 2004-1786	20041202
US	2005181011	A1	20050818	US 2004-1792	20041202
US	2005143817	A1	20050630	US 2004-6899	20041207
US	2005177103	A1	20050811	US 2004-6314	20041207
US	2005177225	A1	20050811	US 2004-6895	20041207
US	2005181004	A1	20050818	US 2004-6289	20041207
US	2006147492	A1	20060706	US 2006-343809	20060131
IN	2006KN01694	A	20070511	IN 2006-KN1694	20060619
IN	2006KN01695	A	20070511	IN 2006-KN1695	20060619
IN	2006KN01698	A	20070511	IN 2006-KN1698	20060619

PRIORITY APPLN. INFO.:

US 2003-523908P	P	20031120
US 2003-524023P	P	20031120
US 2003-525226P	P	20031124
US 2003-526541P	P	20031203
US 2004-578471P	P	20040609
US 2004-586861P	P	20040709
US 2004-986230	A	20041110
US 2004-986231	A	20041110
US 2003-518785P	P	20031110
US 2004-582833P	P	20040624
US 2004-986450	A1	20041110
WO 2004-US37930	W	20041110
WO 2004-US39183	W	20041122
WO 2004-US39346	W	20041122
WO 2004-US39353	W	20041122
WO 2004-US39387	W	20041122

AB Pumps and sensors for contact with tissue are used in combination with an anti-scarring agent (e.g., a cell cycle inhibitor) in order to inhibit scarring that may otherwise occur when the pumps and sensors are implanted within an animal are disclosed. Thus, a drug-coated device was coated with a heparin coating and dipped into a solution of heparin-benzalkonium chloride complex in isopropanol. The device was removed from the solution and air-dried.

L15 ANSWER 9 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:283363 CAPLUS
DOCUMENT NUMBER: 142:329832
TITLE: Combination of a vegf receptor inhibitor with a chemotherapeutic agent
INVENTOR(S): Bold, Guido; Brueggen, Josef Bernhard; Huang, Jerry Min-Jian; Kinder, Frederick Ray, Jr.; Lane, Heidi; Latour, Elisabeth Jeanne; Manley, Paul William; Wood, Jeanette Marjorie
PATENT ASSIGNEE(S): Novartis Ag, Switz.; Novartis Pharma GmbH
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005027972	A2	20050331	WO 2004-EP10686	20040923
WO 2005027972	A3	20051103		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004273615	A1	20050331	AU 2004-273615	20040923
CA 2537991	A1	20050331	CA 2004-2537991	20040923
EP 1682181	A2	20060726	EP 2004-765542	20040923
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
CN 1856327	A	20061101	CN 2004-80027544	20040923
BR 2004014698	A	20061128	BR 2004-14698	20040923
JP 2007505938	T	20070315	JP 2006-527348	20040923
MX 2006PA03163	A	20060605	MX 2006-PA3163	20060320
IN 2006CN00982	A	20070615	IN 2006-CN982	20060322
NO 2006001777	A	20060623	NO 2006-1777	20060421
PRIORITY APPLN. INFO.:			US 2003-505250P P	20030923
			WO 2004-EP10686 W	20040923

OTHER SOURCE(S): MARPAT 142:329832
AB The present invention relates to a combination therapy for treating patients suffering from proliferative diseases or diseases associated with persistent angiogenesis. The patient is treated with: (a) a VEGF inhibitor compound; and (b) one or more chemotherapeutic agents selected from the group consisting of: an aromatase inhibitor; an anti-estrogen, an anti-androgen (especially in the case of prostate cancer) or a gonadorelin agonist; a topoisomerase I inhibitor or a topoisomerase II inhibitor; a microtubule active agent, an alkylating agent, an anti-neoplastic

anti-metabolite or a platin compound; a compound targeting/decreasing a protein or lipid kinase activity or a protein or lipid phosphatase activity, a further anti-angiogenic compound or a compound which induces cell differentiation processes. The patient is treated with :(a) a VEGF inhibitor compound;and (b) one or more chemotherapeutic agents selected from the group consisting of : a bradykinin 1 receptor or an angiotensin II antagonist ; a cyclooxygenase inhibitor , a bisphosphonate , a heparanase inhibitor (prevents heparan sulfate degradation) , e.g. , PI-88 , a biol. response modifier, preferably a lymphokine or interferons , e.g., interferon γ, an ubiquitination inhibitor, or an inhibitor which blocks anti-apoptotic pathways ; an inhibitor of Ras oncogenic isoforms or a farnesyl transferase inhibitor ; a telomerase inhibitor , e.g. , telomestatin ; a protease inhibitor, a matrix metalloproteinase inhibitor , a methionine aminopeptidase inhibitor , e.g. , bengamide or a derivative thereof , or a proteasome inhibitor , e. g. , PS-341. The patient is treated with : (a) a VEGF inhibitor compound (b) one or more chemotherapeutic agents selected from the group consisting of : agents used in the treatment of hematol. malignancies or FMS-like tyrosine kinase inhibitors ; an HSP90 inhibitors ; HDAC inhibitors ; mTOR inhibitors ; somatostatin receptor antagonists ; integrin antagonists ; anti-leukemic compds. ; tumor cell damaging approaches such as ionizing radiation EDG binders ; anthranilic acid amide class of kinase inhibitors ; ribonucleotide reductase inhibitors ; S-adenosylmethionine decarboxylase inhibitors ; antibodies against VEGF or VEGFR ; photodynamic therapy ; angiostatic steroids ; implants containing corticosteroids ; AT1 receptor antagonists ; ACE inhibitors.

L15 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:1101434 CAPLUS

DOCUMENT NUMBER: 142:235549

TITLE: Heat shock-induced matrix metalloproteinase (MMP)-1 and MMP-3 are mediated through ERK and JNK activation and via an autocrine interleukin-6 loop

AUTHOR(S): Park, Chi-Hyun; Lee, Min Jung; Ahn, Jungmi; Kim, Sangmin; Kim, Hyeon Ho; Kim, Kyu Han; Eun, Hee Chul; Chung, Jin Ho

CORPORATE SOURCE: Department of Dermatology, Seoul National University College of Medicine and Laboratory of Cutaneous Aging Research, Clinical Research Institute, Seoul National University Hospital, Seoul, S. Korea

SOURCE: Journal of Investigative Dermatology (2004), 123(6), 1012-1019
CODEN: JIDEAE; ISSN: 0022-202X

PUBLISHER: Blackwell Publishing, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Although many studies have been performed to elucidate the mol. consequences of UV irradiation, little is known about the effect of IR radiation on skin aging. In addition to photons, heat is likely to be generated as a consequence of IR irradiation, and heat shock is widely considered to be an environmental stress. Here we investigated the effect of heat shock on the expressions of matrix metalloproteinase (MMP)-1, MMP-2, and MMP-3 in cultured human skin fibroblasts. Heat shock induced the expression of MMP-1 and MMP-3, but not MMP-2, at the mRNA and protein levels in a temperature-dependent manner, and caused the rapid activation of three distinct mitogen-activated protein kinases (MAPK), extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK. The heat shock-induced MMP-1 and MMP-3 expression was suppressed by the inhibition of ERK and JNK but not by p38 MAPK inhibition. Furthermore, heat shock

increased the synthesis and release of interleukin-6 (IL-6) into culture media. The specific inhibition of IL-6 using a monoclonal antibody against IL-6 greatly reduced the expression of MMP-1 and MMP-3 induced by heat shock. Taken together, our results suggest that ERK and JNK play an important role in the induction of MMP-1 and MMP-3 by heat shock and that the heat shock -induced expression of MMP-1 and MMP-3 is mediated via an IL-6-dependent autocrine mechanism.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:965067 CAPLUS
DOCUMENT NUMBER: 141:406039
TITLE: Combinations for the treatment of diseases involving cell proliferation, migration or apoptosis of myeloma cells, or angiogenesis
INVENTOR(S): Hilberg, Frank; Solca, Flavio; Stefanic, Martin Friedrich; Baum, Anke; Munzert, Gerd; Van Meel, Jacobus C. A.
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE: PCT Int. Appl., 101 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004096224	A2	20041111	WO 2004-EP4363	20040424
WO 2004096224	A3	20041216		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1473043	A1	20041103	EP 2003-9587	20030429
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
AU 2004233576	A1	20041111	AU 2004-233576	20040424
CA 2523868	A1	20041111	CA 2004-2523868	20040424
EP 1622619	A2	20060208	EP 2004-729366	20040424
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
BR 2004009919	A	20060425	BR 2004-9919	20040424
JP 2006524634	T	20061102	JP 2006-500099	20040424
MX 2005PA11656	A	20051215	MX 2005-PA11656	20051028
NO 2005005605	A	20051128	NO 2005-5605	20051128

PRIORITY APPLN. INFO.:

EP 2003-9587	A	20030429
EP 2004-508	A	20040113
EP 2004-1171	A	20040121
WO 2004-EP4363	W	20040424

AB The present invention relates to a pharmaceutical combination for the treatment of diseases which involves cell proliferation, migration or

apoptosis of myeloma cells, or angiogenesis. The invention also relates to a method for the treatment of said diseases, comprising co-administration of effective amts. of specific active compds. and/or co-treatment with radiation therapy, in a ratio which provides an additive and synergistic effect, and to the combined use of these specific compds. and/or radiotherapy for the manufacture of corresponding pharmaceutical combination prepns. The pharmaceutical combination can include selected protein tyrosine kinase receptor antagonists and further chemotherapeutic or naturally occurring semisynthetic or synthetic agents.

L15 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:554409 CAPLUS
DOCUMENT NUMBER: 142:193398
TITLE: Gene expression profiling of in vivo UVB-irradiated human epidermis
AUTHOR(S): Enk, Claes D.; Shahar, Iris; Amariglio, Ninette; Rechavi, Gideon; Kaminski, Naftali; Hochberg, Malka
CORPORATE SOURCE: Department of Dermatolology, The Hadassah-Hebrew University Medical Center, Jerusalem, Israel
SOURCE: Photodermatology, Photoimmunology & Photomedicine (2004), 20(3), 129-137
CODEN: PPPHEW; ISSN: 0905-4383
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background: Several recent studies have employed microarray profiling to study UVB-regulated gene expression in human skin. These studies are all based on UV-irradiated cultured cells that differ substantially from the intact tissues they are supposed to imitate. The purpose of the present study was to analyze the differential expression of UVB-regulated genes in intact human epidermis following in vivo UV irradiation Methods: The forearms of human volunteers were exposed to 4 MED of UVB in vivo, followed by removal of epidermal samples from exposed and non-exposed areas after 24 h. RNA samples were analyzed using oligonucleotide microarray (Affymetrix) technol. analyzing 12 500 genes simultaneously. Verification of selected genes was performed by semi-quant. reverse transcriptase polymerase chain reaction. Results: Gene expression patterns clearly distinguished UV-exposed epidermis from unexposed skin. Classification of these genes into functional categories revealed that several biol. processes are globally affected by UVB. Significant changes were seen in more than 800 genes. Conclusion: Human intact epidermis responds to a single low dose of in vivo UVB irradiation by differential regulation of numerous genes. Our results illustrate the power of global gene expression anal. of human epidermis to identify mol. pathways involved in UV-induced photodamage.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 13 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:308529 CAPLUS
DOCUMENT NUMBER: 140:333599
TITLE: Gene expression profile of human and mouse genes in atopic dermatitis and psoriasis patients and its use for diagnosis, therapy, and drug screening
INVENTOR(S): Itoh, Mikito; Ogawa, Kaoru; Shinagawa, Akira; Sudo, Hajime; Ogawa, Hideoki; Ra, Chisei; Mitsuishi, Kouichi
PATENT ASSIGNEE(S): Genox Research, Inc., Japan; Juntendo University
SOURCE: PCT Int. Appl., 611 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004031386	A1	20040415	WO 2003-JP9808	20030801
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003252326	A1	20040423	AU 2003-252326	20030801
PRIORITY APPLN. INFO.:			JP 2002-229318 A	20020806
			JP 2003-136543 A	20030514
			WO 2003-JP9808 W	20030801

AB This invention provides gene expression profile between a rash site and a no-rash site in a patient with atopic dermatitis or a patient with psoriasis. The invention also provides gene expression profile between a no-rash site in such a disease and a normal subject. Animal models, particularly mouse for those diseases are also claimed. The gene expression profile provided in this invention can be used for diagnosis, therapy, and drug screening for atopic dermatitis and psoriasis.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS
DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606

PRIORITY APPLN. INFO.: GB 2002-5537 A 20020308
GB 2002-29054 A 20021212
WO 2003-EP2365 W 20030307
OTHER SOURCE(S): MARPAT 139:240351
AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:48083 CAPLUS
DOCUMENT NUMBER: 138:333688
TITLE: Infrared-A radiation-induced matrix metalloproteinase 1 expression is mediated through extracellular signal-regulated kinase 1/2 activation in human dermal fibroblasts
AUTHOR(S): Schieke, Stefan M.; Stege, Helger; Kurten, Viola; Grether-Beck, Susanne; Sics, Helmut; Krutmann, Jean
CORPORATE SOURCE: Institut fur Umweltmedizinische Forschung (IUF) an der Heinrich-Heine-Universitat GmbH, Duesseldorf, D-40225, Germany
SOURCE: Journal of Investigative Dermatology (2002), 119(6), 1323-1329
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In addition to UV radiation, human skin is exposed to IR radiation from natural sunlight as well as artificial UV and IR irradiation devices used for therapeutic or cosmetic purposes. The mol. consequences resulting from IR exposure are virtually unknown. In this study we have investigated whether IR has the capacity to affect gene expression in human skin cells. Exposure of cultured human dermal fibroblasts to IR in the range of 760-1400 nm (IR-A) induced the expression of matrix metalloproteinase 1 at the mRNA and protein level in a time- and concentration-dependent manner. Expression of tissue inhibitor of matrix metalloproteinase 1 remained unaltered. These effects were not mediated by the generation of heat by IR-A. Furthermore, IR-A did not induce heat shock protein 70 expression in human dermal fibroblasts under conditions that increased matrix metalloproteinase 1 expression. Here we provide evidence that IR-A activated mitogen-activated protein kinase pathways. Extracellular signal-regulated kinase 1/2 and p38-mitogen-activated protein kinase were rapidly activated after IR-A exposure. The mitogen-activated protein kinase/extracellular signal-regulated kinase inhibitor PD 98059, which specifically blocked the extracellular signal-regulated kinase pathway, prevented IR-A-induced matrix metalloproteinase 1 expression. Upregulation of matrix metalloproteinase 1 expression by IR-A was thus shown to be dependent on extracellular signal-regulated kinase 1/2 activation. In conclusion, this study demonstrates that IR-A is capable of inducing matrix metalloproteinase 1 expression in human dermal fibroblasts via activation of the extracellular signal-regulated kinase 1/2 signaling pathway. This previously unrecognized property of IR-A points to its possible role in the photoaging of human skin.
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L16          35 L13 AND (RADIOTHER? OR RADIATION OR CHEMORADIOTHERAPY)

=> dup rem
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PROCESSING COMPLETED FOR L16
L17          27 DUP REM L16 (8 DUPLICATES REMOVED)

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L17  ANSWER 1 OF 27  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:         2007:433685  CAPLUS
DOCUMENT NUMBER:          146:460567
TITLE:                    Nucleic acid vaccines encoding matrix
                          metalloproteinase 11 and immunoenhancing
                          element against cancer or carcinoma
INVENTOR(S):              Aurisicchio, Luigi; La Monica, Nicola
PATENT ASSIGNEE(S):       Istituto di Ricerche di Biologia Molecolare P.
                          Angeletti S.p.A., Italy; Ciliberto, Gennaro; Lazzaro,
                          Domenico; Mori, Federica; Peruzzi, Daniela
SOURCE:                   PCT Int. Appl., 68pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
     WO 2007042169        A2    20070419    WO 2006-EP9536          20061003
     WO 2007042169        A3    20070531
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
             KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
             MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS,
             RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
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CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: US 2005-724498P P 20051007

AB Compns. comprising matrix metalloproteinase 11 (MMP-11) or stromelysin-3 (ST-3) or the nucleic acid encoding the MMP-11 for use in vaccines for treating tumors and cancers, which overexpress MMP-11, are described. In particular embodiments, the compns. comprise a nucleic acid encoding a fusion polypeptide that includes the catalytically inactivated MMP-11 linked at the C-terminus to an immunoenhancing element wherein the codons encoding the MMP-11 and the immunoenhancing element have been optimized for enhanced expression of the fusion polypeptide in human cells. In other embodiments, the compns. comprise the catalytically inactivated MMP-11 linked at the C-terminus to an immunoenhancing element. The compns. can be used alone or in synergy with vaccines against other tumor associated antigens as well as with conventional therapies such as radiation therapy and chemotherapy.

L17 ANSWER 2 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2007096345 EMBASE

TITLE: Recent advances in the management of osteosarcoma and forthcoming therapeutic strategies.

AUTHOR: Lamoureux F.; Trichet V.; Chipoy C.; Blanchard F.; Gouin F.; Redini F.

CORPORATE SOURCE: Dr. F. Redini, Universite de Nantes, Physiopathologie de la Resorption Osseuse et Therapie des Tumeurs Osseuses Primitives, Faculte de Medecine, 1 rue Gaston Veil, 44035 Nantes Cedex 1, France. francoise.redini@univ-nantes.fr

SOURCE: Expert Review of Anticancer Therapy, (Feb 2007) Vol. 7, No. 2, pp. 169-181.
Refs: 88
ISSN: 1473-7140 E-ISSN: 1744-8328 CODEN: ERATBJ

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
030 Clinical and Experimental Pharmacology
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 2 Apr 2007
Last Updated on STN: 2 Apr 2007

AB Osteosarcoma is the most frequent primary bone tumor and occurs mainly in young patients (average age: 18 years). No evolution of the survival rates has been recorded for two decades in response to current treatment, associating often toxic and badly tolerated cures of chemotherapy (given a significant rate of bad responders) with preserving surgery. Among the proposed innovative strategies, immune-based therapy, antiangiogenesis agents, tumor-suppressor or suicide gone therapy, or anticancer drugs not commonly used in osteosarcoma are presented. A further strategy is to target the tumor microenvironment rather than the tumor itself. .COPYRGT. 2007 Future Drugs Ltd.

L17 ANSWER 3 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:1157631 CAPLUS

DOCUMENT NUMBER: 145:483673

TITLE: Novel methods and devices for evaluating poisons

INVENTOR(S): Ching, Edwin P.; Johnson, Dale E.; Sudarsanam, Sucha

PATENT ASSIGNEE(S): Emiliem, USA

SOURCE: PCT Int. Appl., 132pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006116622	A2	20061102	WO 2006-US16067	20060426
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006253262	A1	20061109	US 2006-380388	20060426
EP 1880332	A2	20080123	EP 2006-751675	20060426
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				

PRIORITY APPLN. INFO.:		
US 2005-675741P	P	20050427
US 2006-778133P	P	20060301
WO 2006-US16067	W	20060426

AB Methods and devices useful for evaluating poisons or other chemical entities, and for using such methods to forecast unfavorable drug effects. The present invention provides lists of biomarkers for anal., either directly or indirectly, which affect the toxicity pathways. These may be evaluated at many levels, including genetic, genotyping, evaluation of combination pairing of diploid alleles or haplotypes, RNA expression, protein expression, functional activity, posttranslational anal. or evaluation, etc. Thus, the biomarkers refer to the corresponding genetic information, RNA, protein, or other structural embodiments thereof. And the means to use these biomarkers, e.g., to evaluate status of toxicity pathways, to evaluate individual risk or susceptibility to various toxic pathways from exposure or therapeutic intervention, to generate test systems for drug development, are all provided by identifying critical and significant contributors to the pathway progression. The present invention is directed to accelerating the speed of development and reducing the resource investment necessary to determine these features for directing use of such substances or treatments to appropriate biol. contexts.

L17 ANSWER 4 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:167588 CAPLUS
DOCUMENT NUMBER: 144:254148
TITLE: Aminopteridinones as anticancer agents, their preparation, pharmaceutical compositions, and use in therapy
INVENTOR(S): Munzert, Gerd; Steegmaier, Martin; Baum, Anke
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE: PCT Int. Appl., 158 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2006018182	A1	20060223	WO 2005-EP8623		20050809
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
US 2006058311	A1	20060316	US 2005-189540		20050726
AU 2005274384	A1	20060223	AU 2005-274384		20050809
CA 2576269	A1	20060223	CA 2005-2576269		20050809
EP 1827441	A1	20070905	EP 2005-770228		20050809
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BA, HR, YU					
CN 101039673	A	20070919	CN 2005-80035272		20050809
IN 2007DN00888	A	20070803	IN 2007-DN888		20070202
MX 200701853	A	20070328	MX 2007-1853		20070214
KR 2007050478	A	20070515	KR 2007-705955		20070314
PRIORITY APPLN. INFO.:			EP 2004-19361	A	20040814
			EP 2004-19448	A	20040817
			WO 2005-EP8623	W	20050809
OTHER SOURCE(S):	MARPAT 144:254148				

GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The invention relates to a group of aminopteridinones I, which are useful for the treatment of diseases which involve cell proliferation. In compds. I, R1 and R2 are independently selected from H and (un)substituted C1-6 alkyl, or R1 and R2 together form a 2- to 5-membered alkylene bridge, optionally containing 1 or 2 heteroatoms; R3 is (un)substituted C1-12 alkyl, C2-12 alkenyl, C2-12 alkynyl, C6-14 aryl, etc.; R4 is H, OH, CN, halo, (un)substituted amino, (un)substituted C1-6 alkyl, C1-5 alkoxy, etc.; L is (un)substituted C2-10 alkylene, (un)substituted C2-10 alkenylene, (un)substituted C6-14 arylene, etc.; R5 is (un)substituted morpholinyl, (un)substituted piperidinyl, (un)substituted piperazinyl, (un)substituted piperazinylcarbonyl, (un)substituted pyrrolidinyl, (un)substituted thiomorpholinyl, etc.; n is 0 or 1; and m is 1 or 2; including tautomers, stereoisomers, salts, solvates, polymorphs, and prodrugs thereof. The invention also relates to the preparation of I, pharmaceutical compns. comprising a compound I, at least one other therapeutic agent, optionally with one or more pharmaceutically acceptable excipients, as well as to the use of the compns. for the treatment of diseases which involve cell proliferation, migration or apoptosis of cancer cells, or angiogenesis. Esterification of (R)-2-aminobutyric acid and reductive condensation with cyclopentanone gave cyclopentylamine II, which underwent regioselective substitution of 2,4-dichloro-5-nitropyrimidine and reductive heterocyclization to form pteridinone III. N-Methylation of III followed by substitution with 4-amino-3-methoxybenzoic acid and amidation with

1-methyl-4-aminopiperidine resulted in the formation of aminopteridinone IV. A combination of suboptimal doses of irinotecan and compound IV shows an additive/synergistic effect in a human colon carcinoma model and is well tolerated. Meanwhile, compound IV acts at least additively with docetaxel in a human non-small cell lung carcinoma model and not antagonistically with gemcitabine in a human adenocarcinoma model.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 5 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:120414 CAPLUS
DOCUMENT NUMBER: 144:184702
TITLE: Gene expression profiles for identifying patients at risk of developing encephalitis following immunotherapy for Alzheimer's disease
INVENTOR(S): O'Toole, Margot; Dorner, Andrew J.; Janszen, Derek B.; Slonim, Donna K.; Mounts, William M.; Reddy, Padmalatha S.; Hill, Andrew A.
PATENT ASSIGNEE(S): Wyeth, USA
SOURCE: PCT Int. Appl., 298 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006014755	A2	20060209	WO 2005-US25771	20050720
WO 2006014755	A3	20060413		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2571856	A1	20060209	CA 2005-2571856	20050720
US 2006073496	A1	20060406	US 2005-186236	20050720
EP 1784509	A2	20070516	EP 2005-795582	20050720
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

PRIORITY APPLN. INFO.: US 2004-589877P P 20040720
US 2005-672716P P 20050418
WO 2005-US25771 W 20050720

AB The present invention generally relates to a method for an improved treatment for Alzheimer's disease (AD) using immunotherapy, e.g., immunotherapy targeting β amyloid (Aβ) and immunotherapy based on AN1792. By ANOVA and GeneCluster analyses of Affymetrix U133A GeneChip data, statistically significant assocns. were detected between the gene expression profiles of peripheral blood mononuclear cells of patients prior to immunization with AN1792 and the post-immunization development of encephalitis. In addition, statistically significant assocns. were found between the pre-immunization gene expression profile in PBMCs and post-immunization development of IgG response. The method allows for predicting an adverse clin. response, and therefore allows for an improved safety profile of AN1792. In another embodiment, the method allows for

predicting a favorable clin. response, and therefore allows for an improved efficacy profile of AN1792. The methods of the present invention may be combined to predict a favorable clin. response and the lack of an adverse clin. response.

L17 ANSWER 6 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006586498 EMBASE
TITLE: Nitric oxide and the regulation of apoptosis in tumour cells.
AUTHOR: Tarr J.M.; Eggleton P.; Winyard P.G.
CORPORATE SOURCE: P.G. Winyard, Institute of Biomedical and Clinical Science, Peninsula Medical School, Universities of Exeter and Plymouth, Exeter, Devon EX1 2LU, United Kingdom. paul.winyard@pms.ac.uk
SOURCE: Current Pharmaceutical Design, (Dec 2006) Vol. 12, No. 34, pp. 4445-4468.
Refs: 313
ISSN: 1381-6128 CODEN: CPDEFP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jan 2007
Last Updated on STN: 24 Jan 2007

AB Nitric oxide (NO) is a small, highly reactive, diffusible free radical which has been implicated in many physiological and pathophysiological processes. It has either pro-apoptotic or anti-apoptotic effects on cells, depending upon a host of factors. This review outlines some of the regulatory molecules and organelles involved in the apoptotic pathways that can be influenced by the presence of NO, including p53, Bcl-2, caspases, mitochondria, and heat shock proteins. The effects of NO on the apoptosis of tumour cells are also examined. .COPYRGT. 2006 Bentham Science Publishers Ltd.

L17 ANSWER 7 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2006:151685 CAPLUS
DOCUMENT NUMBER: 144:309813
TITLE: The receptor for advanced glycation end products is highly expressed in the skin and upregulated by advanced glycation end products and tumor necrosis factor-alpha
AUTHOR(S): Lohwasser, Christina; Neureiter, Daniel; Weigle, Bernd; Kirchner, Thomas; Schuppan, Detlef
CORPORATE SOURCE: Department of Medicine I, University of Erlangen-Nuernberg, Germany
SOURCE: Journal of Investigative Dermatology (2006), 126(2), 291-299
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Advanced glycation end products (AGEs) form non-enzymically from reactions of proteins with reducing sugars. In the skin, AGEs were reported to accumulate in dermal elastin and collagens and to interact nonspecifically with the cell membrane of dermal fibroblasts. Therefore, AGEs may influence the process of skin aging. We investigated the presence of the

AGE receptor RAGE in skin and the influence of AGEs on receptor expression and the formation of extracellular matrix (ECM). Sections of sun-protected and sun-exposed skin were analyzed with monoclonal antibodies against (RAGE), heat-shock protein 47, factor XIIIa, CD31, and CD45. RAGE was mainly expressed in fibroblasts, dendrocytes, and keratinocytes and to a minor extent in endothelial and mononuclear cells. Human foreskin fibroblasts (HFFs) highly expressed RAGE on the protein and mRNA level when analyzed by quant. Western blotting and real-time PCR. Incubation of HFFs with the specific RAGE ligand Nε-(carboxymethyl)lysine-modified BSA (CML-BSA) and tumor necrosis factor-alpha resulted in significant upregulation of RAGE expression. CML-BSA induced a mildly profibrogenic pattern, increasing connective tissue growth factor, transforming growth factor-beta (TGF-β) 1, and procollagen-α1(I) mRNA, whereas expression of matrix metalloproteinase (MMP)-1, -2, -3, and -12 was unaffected. We conclude that in HFFs, AGE-RAGE interactions may influence the process of skin aging through mild stimulation of ECM gene expression.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 8 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:816927 CAPLUS
DOCUMENT NUMBER: 145:267789
TITLE: The dynamic phase of cancer cells by the low temperature narrow wavelength far infrared radiation
AUTHOR(S): Hosokawa, Hiroyoshi
CORPORATE SOURCE: Dep. Oral Maxill. Surg., Grad. Sch. Dentistry, the University of Tokuyama, Japan
SOURCE: Shikoku Shigakkai Zasshi (2006), 19(1), 35-54 CODEN: SSZAED; ISSN: 0914-6091
PUBLISHER: Shikoku Shigakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Far IR ray (FIR) are known to have some effects on the human body, but little is known about the non-fever effects in normal thermal fields. We developed a CO2 incubator and an animal raiser that is able to radiate low temperature narrow wavelength (limited) FIR at wavelength of 4 to 20 μm with a peak wavelength 7 to 12 μm, which had strong effects on living tissue, and we investigated the effects of this FIR on cancer cells. In in vitro analyses, analyses of cell proliferation and cell cycle were carried out using 5-bromo-2'-deoxy-uridine (BrdU) incorporation and flow cytometry in three cancer cell lines: the human vulval epidermal cell line A431, the human tongue squamous cell carcinoma (SCC) line HSC3, and the human gingiva SCC line Sa3. In addition, from the viewpoint of the heat shock proteins (HSPs), especially the HSP70 protein, having cytoprotection for various stresses, Hsp70A gene expression was examined using real-time reverse transcription polymerase chain reaction. The effect of HSP70 protein on cell proliferation for limited FIR was analyzed by transfecting Hsp70A expression vector or by repressing Hsp70A and Hsp70C mRNA using gene silencing methods with siRNA. In in vivo analyses, we generated xenograft tumors of A431 and Sa3 cells and examined the changes of tumor volume, genetic alteration and histol. observation. As a result, limited FIR suppressed cell proliferation of HSC3 and Sa3 cells, not A431 cells. The cell cycle of HSC3 cells was mainly delayed by limited FIR in the G2/M stage, while necrotic cells of Sa3 cells slightly increased by limited FIR. Moreover, the expression of Hsp70A gene and HSP70 protein was higher on A431 cells whose cell proliferation was not suppressed by limited FIR. On BrdU incorporation anal. under the condition in which HSP70 protein was repressed, BrdU incorporation of A431 cells was suppressed. In in vivo analyses, limited FIR suppressed both the growth of A431 tumor and Sa3 tumor. Tumor tissues of A431 in limited

FIR group were encapsulated and matrix metalloproteinase (MMP)-1, -9, -10, -13 were significantly suppressed in the protein level. On the other hand, limited FIR induced the apoptosis in the Sa3 tumor. These findings in vitro suggest that limited FIR suppressed the proliferation of certain cancer cells, and the suppressive effect depended on expression level of HSP70 protein. These findings in vivo that limited FIR suppressed the tumor growth of A431 by inhibiting MMPs, and that of Sa3 by inducing apoptosis.

L17 ANSWER 9 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:493568 CAPLUS
DOCUMENT NUMBER: 143:48169
TITLE: Implantable sensors and pumps and anti-scarring agents
INVENTOR(S): Hunter, William L.; Gravett, David M.; Toleikis, Philip M.; Maiti, Arpita
PATENT ASSIGNEE(S): Angiotech International A.-G., Switz.
SOURCE: PCT Int. Appl., 1619 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 19
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005051871	A2	20050609	WO 2004-US39387	20041122
WO 2005051871	A9	20060727		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005148512	A1	20050707	US 2004-986230	20041110
US 2005181977	A1	20050818	US 2004-986231	20041110
CN 101094613	A	20071226	CN 2004-80031664	20041110
AU 2004293463	A1	20050609	AU 2004-293463	20041122
CA 2536242	A1	20050609	CA 2004-2536242	20041122
WO 2005051232	A2	20050609	WO 2004-US39346	20041122
WO 2005051232	A3	20051208		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2006055008	A2	20060526	WO 2004-US39353	20041122
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,				

TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

EP 1685085	A2	20060802	EP 2004-817879		20041122
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS					
CN 1878514	A	20061213	CN 2004-80033341		20041122
JP 2007513650	T	20070531	JP 2006-541669		20041122
US 2005149158	A1	20050707	US 2004-409		20041129
US 2005175662	A1	20050811	US 2004-451		20041129
US 2005175661	A1	20050811	US 2004-999205		20041129
US 2005186243	A1	20050825	US 2004-97		20041129
US 2005186242	A1	20050825	US 2004-999204		20041129
US 2005191331	A1	20050901	US 2004-1419		20041130
US 2005175663	A1	20050811	US 2004-1791		20041202
US 2005181008	A1	20050818	US 2004-1786		20041202
US 2005181011	A1	20050818	US 2004-1792		20041202
US 2005143817	A1	20050630	US 2004-6899		20041207
US 2005177103	A1	20050811	US 2004-6314		20041207
US 2005177225	A1	20050811	US 2004-6895		20041207
US 2005181004	A1	20050818	US 2004-6289		20041207
US 2006147492	A1	20060706	US 2006-343809		20060131
IN 2006KN01694	A	20070511	IN 2006-KN1694		20060619
IN 2006KN01695	A	20070511	IN 2006-KN1695		20060619
IN 2006KN01698	A	20070511	IN 2006-KN1698		20060619
PRIORITY APPLN. INFO.:			US 2003-523908P	P	20031120
			US 2003-524023P	P	20031120
			US 2003-525226P	P	20031124
			US 2003-526541P	P	20031203
			US 2004-578471P	P	20040609
			US 2004-586861P	P	20040709
			US 2004-986230	A	20041110
			US 2004-986231	A	20041110
			US 2003-518785P	P	20031110
			US 2004-582833P	P	20040624
			US 2004-986450	A1	20041110
			WO 2004-US37930	W	20041110
			WO 2004-US39183	W	20041122
			WO 2004-US39346	W	20041122
			WO 2004-US39353	W	20041122
			WO 2004-US39387	W	20041122

AB Pumps and sensors for contact with tissue are used in combination with an anti-scarring agent (e.g., a cell cycle inhibitor) in order to inhibit scarring that may otherwise occur when the pumps and sensors are implanted within an animal are disclosed. Thus, a drug-coated device was coated with a heparin coating and dipped into a solution of heparin-benzalkonium chloride complex in isopropanol. The device was removed from the solution and air-dried.

L17 ANSWER 10 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER:	2005:283363 CAPLUS
DOCUMENT NUMBER:	142:329832
TITLE:	Combination of a vegf receptor inhibitor with a chemotherapeutic agent
INVENTOR(S):	Bold, Guido; Brueggen, Josef Bernhard; Huang, Jerry Min-Jian; Kinder, Frederick Ray, Jr.; Lane, Heidi; Latour, Elisabeth Jeanne; Manley, Paul William; Wood, Jeanette Marjorie
PATENT ASSIGNEE(S):	Novartis Ag, Switz.; Novartis Pharma GmbH

SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005027972	A2	20050331	WO 2004-EP10686	20040923
WO 2005027972	A3	20051103		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004273615	A1	20050331	AU 2004-273615	20040923
CA 2537991	A1	20050331	CA 2004-2537991	20040923
EP 1682181	A2	20060726	EP 2004-765542	20040923
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
CN 1856327	A	20061101	CN 2004-80027544	20040923
BR 2004014698	A	20061128	BR 2004-14698	20040923
JP 2007505938	T	20070315	JP 2006-527348	20040923
MX 2006PA03163	A	20060605	MX 2006-PA3163	20060320
IN 2006CN00982	A	20070615	IN 2006-CN982	20060322
NO 2006001777	A	20060623	NO 2006-1777	20060421
PRIORITY APPLN. INFO.:			US 2003-505250P P	20030923
			WO 2004-EP10686 W	20040923

OTHER SOURCE(S): MARPAT 142:329832

AB The present invention relates to a combination therapy for treating patients suffering from proliferative diseases or diseases associated with persistent angiogenesis. The patient is treated with: (a) a VEGF inhibitor compound; and (b) one or more chemotherapeutic agents selected from the group consisting of: an aromatase inhibitor; an anti-estrogen, an anti-androgen (especially in the case of prostate cancer) or a gonadorelin agonist; a topoisomerase I inhibitor or a topoisomerase II inhibitor; a microtubule active agent, an alkylating agent, an anti-neoplastic anti-metabolite or a platin compound; a compound targeting/decreasing a protein or lipid kinase activity or a protein or lipid phosphatase activity, a further anti-angiogenic compound or a compound which induces cell differentiation processes. The patient is treated with :(a) a VEGF inhibitor compound;and (b) one or more chemotherapeutic agents selected from the group consisting of : a bradykinin 1 receptor or an angiotensin II antagonist ; a cyclooxygenase inhibitor , a bisphosphonate , a heparanase inhibitor (prevents heparan sulfate degradation) , e.g. , PI-88 , a biol. response modifier, preferably a lymphokine or interferons , e.g., interferon γ, an ubiquitination inhibitor, or an inhibitor which blocks anti-apoptotic pathways ; an inhibitor of Ras oncogenic isoforms or a farnesyl transferase inhibitor ; a telomerase inhibitor , e.g. , telomestatin ; a protease inhibitor, a matrix metalloproteinase inhibitor , a methionine aminopeptidase inhibitor , e.g. , bengamide or a derivative thereof , or a proteasome inhibitor , e. g. , PS-341. The patient is treated with : (a) a VEGF inhibitor compound (b) one or more chemotherapeutic agents selected from the group consisting of : agents used in the treatment of hematol. malignancies or FMS-like tyrosine kinase

inhibitors ; an HSP90 inhibitors ; HDAC inhibitors ; mTOR inhibitors ; somatostatin receptor antagonists ; integrin antagonists ; anti-leukemic compds. ; tumor cell damaging approaches such as ionizing radiation EDG binders ; anthranilic acid amide class of kinase inhibitors ; ribonucleotide reductase inhibitors ; S-adenosylmethionine decarboxylase inhibitors ; antibodies against VEGF or VEGFR ; photodynamic therapy ; angiostatic steroids ; implants containing corticosteroids ; AT1 receptor antagonists ; ACE inhibitors.

L17 ANSWER 11 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:1293778 CAPLUS
DOCUMENT NUMBER: 144:35066
TITLE: Gene expression profiling in the prostate in the diagnosis and Gleason staging of high- and low-grade tumors
INVENTOR(S): Shekar, Mamatha; Zhang, Zhaomei; Caldwell, Mitchell C.; Chen, Zuxiong; Fan, Zhenbin; McNeal, John E.; Nolley, Rosalie; Stamey, Thomas A.; Warrington, Janet A.; Palma, John F.
PATENT ASSIGNEE(S): Affymetrix, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 411,537.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2005272052	A1	20051208	US 2004-975592		20041027
US 2004029151	A1	20040212	US 2003-411537		20030409
PRIORITY APPLN. INFO.:			US 2002-371304P	P	20020409
			US 2003-411537	A2	20030409

AB Many genes are affected in prostate cancers which have not been previously identified. This includes genes that have been up-regulated or down-regulated. Monitoring the expression levels of these genes is useful to identify the existence of prostate cancer and to differentiate low-risk (Gleason grade 3), and high risk (Gleason grade 4 or 5) tumors. Also, monitoring the expression levels of these genes is useful to predict the effectiveness of treatment, outcome, use of therapeutics, and screening drugs useful for the treatment of prostate cancer.

L17 ANSWER 12 OF 27 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 1
ACCESSION NUMBER: 2005:362767 BIOSIS
DOCUMENT NUMBER: PREV200510154562
TITLE: Novel aspects of intrinsic and extrinsic aging of human skin: Beneficial effects of soy extract.
AUTHOR(S): Suedel, Kirstin M.; Venzke, Kirsten; Mielke, Heiko; Breitenbach, Ute; Mundt, Claudia; Jaspers, Soeren; Koop, Urte; Sauermann, Kirsten; Knussmann-Hartig, Elke; Moll, Ingrid; Gercken, Guenther; Young, Antony R.; Staeb, Franz; Wenck, Horst; Gallinat, Stefan [Reprint Author]
CORPORATE SOURCE: Beiersdorf AG, Paul Gerson Unna Skin Res Ctr, Unnastr 48, D-20245 Hamburg, Germany
stefan.gallinat@beiersdorf.com
SOURCE: Photochemistry and Photobiology, (MAY-JUN 2005) Vol. 81, No. 3, pp. 581-587.
CODEN: PHCBAP. ISSN: 0031-8655.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 14 Sep 2005
Last Updated on STN: 14 Sep 2005

AB Biochemical and structural changes of the dermal connective tissue substantially contribute to the phenotype of aging skin. To study connective tissue metabolism with respect to ultraviolet (UV) exposure, we performed an in vitro (human dermal fibroblasts) and an in vivo complementary DNA array study in combination with protein analysis in young and old volunteers. Several genes of the collagen metabolism such as Collagen I, III and VI as well as heat shock protein 47 and matrix metalloproteinase-1 are expressed differentially, indicating UV-mediated effects on collagen expression, processing and degradation. In particular, Collagen I is time and age dependently reduced after a single UV exposure in human skin in vivo. Moreover, older subjects display a lower baseline level and a shorter UV-mediated increase in hyaluronan (HA) levels. To counteract these age-dependent changes, cultured fibroblasts were treated with a specific soy extract. This treatment resulted in increased collagen and HA synthesis. In a placebo-controlled in vivo study, topical application of an isoflavone-containing emulsion significantly enhanced the number of dermal papillae per area after 2 weeks. Because the flattening of the dermal-epidermal junction is the most reproducible structural change in aged skin, this soy extract appears to rejuvenate the structure of mature skin.

L17 ANSWER 13 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:965067 CAPLUS
DOCUMENT NUMBER: 141:406039
TITLE: Combinations for the treatment of diseases involving cell proliferation, migration or apoptosis of myeloma cells, or angiogenesis
INVENTOR(S): Hilberg, Frank; Solca, Flavio; Stefanic, Martin Friedrich; Baum, Anke; Munzert, Gerd; Van Meel, Jacobus C. A.
PATENT ASSIGNEE(S): Boehringer Ingelheim International G.m.b.H., Germany; Boehringer Ingelheim Pharma G.m.b.H. & Co. K.-G.
SOURCE: PCT Int. Appl., 101 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004096224	A2	20041111	WO 2004-EP4363	20040424
WO 2004096224	A3	20041216		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1473043	A1	20041103	EP 2003-9587	20030429
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
AU 2004233576	A1	20041111	AU 2004-233576	20040424
CA 2523868	A1	20041111	CA 2004-2523868	20040424

EP 1622619 A2 20060208 EP 2004-729366 20040424
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
BR 2004009919 A 20060425 BR 2004-9919 20040424
JP 2006524634 T 20061102 JP 2006-500099 20040424
MX 2005PA11656 A 20051215 MX 2005-PA11656 20051028
NO 2005005605 A 20051128 NO 2005-5605 20051128
PRIORITY APPLN. INFO.: EP 2003-9587 A 20030429
EP 2004-508 A 20040113
EP 2004-1171 A 20040121
WO 2004-EP4363 W 20040424

AB The present invention relates to a pharmaceutical combination for the treatment of diseases which involves cell proliferation, migration or apoptosis of myeloma cells, or angiogenesis. The invention also relates to a method for the treatment of said diseases, comprising co-administration of effective amts. of specific active compds. and/or co-treatment with radiation therapy, in a ratio which provides an additive and synergistic effect, and to the combined use of these specific compds. and/or radiotherapy for the manufacture of corresponding pharmaceutical combination prepns. The pharmaceutical combination can include selected protein tyrosine kinase receptor antagonists and further chemotherapeutic or naturally occurring semisynthetic or synthetic agents.

L17 ANSWER 14 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:308529 CAPLUS
DOCUMENT NUMBER: 140:333599
TITLE: Gene expression profile of human and mouse genes in atopic dermatitis and psoriasis patients and its use for diagnosis, therapy, and drug screening
INVENTOR(S): Itoh, Mikito; Ogawa, Kaoru; Shinagawa, Akira; Sudo, Hajime; Ogawa, Hideoki; Ra, Chisei; Mitsuishi, Kouichi
PATENT ASSIGNEE(S): Genox Research, Inc., Japan; Juntendo University
SOURCE: PCT Int. Appl., 611 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004031386	A1	20040415	WO 2003-JP9808	20030801
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003252326	A1	20040423	AU 2003-252326	20030801
PRIORITY APPLN. INFO.:			JP 2002-229318	A 20020806
			JP 2003-136543	A 20030514
			WO 2003-JP9808	W 20030801

AB This invention provides gene expression profile between a rash site and a no-rash site in a patient with atopic dermatitis or a patient with psoriasis. The invention also provides gene expression profile between a no-rash site in such a disease and a normal subject. Animal models, particularly mouse for those diseases are also claimed. The gene

expression profile provided in this invention can be used for diagnosis, therapy, and drug screening for atopic dermatitis and psoriasis.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 15 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 2
ACCESSION NUMBER: 2004518961 EMBASE
TITLE: Heat shock-induced matrix metalloproteinase (MMP)-1 and MMP-3 are mediated through ERK and JNK activation and via an autocrine interleukin-6 loop.
AUTHOR: Park C.-H.; Min J.L.; Ahn J.; Kim S.; Hyeon H.K.; Kyu H.K.; Hee C.E.; Jin H.C.
CORPORATE SOURCE: Dr. H.C. Jin, Department of Dermatology, Seoul National University Hospital, 28 Yongon-dong, Chongno-gu, Seoul, 110-744, Korea, Republic of. jhchung@snu.ac.kr
SOURCE: Journal of Investigative Dermatology, (Dec 2004) Vol. 123, No. 6, pp. 1012-1019.
Refs: 47
ISSN: 0022-202X CODEN: JIDEAE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
029 Clinical and Experimental Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Dec 2004
Last Updated on STN: 28 Dec 2004
AB Although many studies have been performed to elucidate the molecular consequences of ultraviolet irradiation, little is known about the effect of infrared radiation on skin aging. In addition to photons, heat is likely to be generated as a consequence of infrared irradiation, and heat shock is widely considered to be an environmental stress. Here we investigated the effect of heat shock on the expressions of matrix metalloproteinase (MMP)-1, MMP-2, and MMP-3 in cultured human skin fibroblasts. Heat shock induced the expression of MMP-1 and MMP-3, but not MMP-2, at the mRNA and protein levels in a temperature-dependent manner, and caused the rapid activation of three distinct mitogen-activated protein kinases (MAPK), extracelluar signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK. The heat shock-induced MMP-1 and MMP-3 expression was suppressed by the inhibition of ERK and JNK but not by p38 MAPK inhibition. Furthermore, heat shock increased the synthesis and release of interleukin-6 (IL-6) into culture media. The specific inhibition of IL-6 using a monoclonal antibody against IL-6 greatly reduced the expression of MMP-1 and MMP-3 induced by heat shock. Taken together, our results suggest that ERK and JNK play an important role in the induction of MMP-1 and MMP-3 by heat shock and that the heat shock-induced expression of MMP-1 and MMP-3 is mediated via an IL-6-dependent autocrine mechanism.

L17 ANSWER 16 OF 27 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:319395 BIOSIS
DOCUMENT NUMBER: PREV200510114790
TITLE: Heat shock-induced expression of matrix metalloproteinase-1 is mediated by activation of extracellular signal-regulated kinase and c-Jun N-terminal kinase and via an interleukin 6-dependent autocrine

mechanism in human skin fibroblasts.
AUTHOR(S): Park, C. [Reprint Author]; Lee, M.; Ahn, I.; Kim, S.; Shin, M.; Kim, K.; Eun, H.; Chung, I.
CORPORATE SOURCE: Seoul Natl Univ, Dept Dermatol, Seoul Natl Univ Hosp, Lab Cutaneous Aging Res,Clin Res Inst,Coll Med, Seoul 151, South Korea
SOURCE: Journal of Investigative Dermatology, (MAR 2004) Vol. 122, No. 3, pp. A140.
Meeting Info.: 65th Annual Meeting of the Society-for-Investigative-Dermatology. Providence, RI, USA. April 28 -May 01, 2004. Soc Investigat Dermatol.
CODEN: JIDEAE. ISSN: 0022-202X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Aug 2005
Last Updated on STN: 25 Aug 2005

L17 ANSWER 17 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2004293118 EMBASE
TITLE: Immune response against dying tumor cells.
AUTHOR: Zitvogel L.; Casares N.; Pequignot M.O.; Chaput N.; Albert M.L.; Kroemer G.
CORPORATE SOURCE: Institut Gustave Roussy Villejuif, France
SOURCE: Advances in Immunology, (2004) Vol. 84, pp. 131-179.
Refs: 246
ISSN: 0065-2776 CODEN: ADIMAV
PUBLISHER IDENT.: S 0065-2776(04)84004-5
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Aug 2004
Last Updated on STN: 5 Aug 2004

L17 ANSWER 18 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:554409 CAPLUS
DOCUMENT NUMBER: 142:193398
TITLE: Gene expression profiling of in vivo UVB-irradiated human epidermis
AUTHOR(S): Enk, Claes D.; Shahar, Iris; Amariglio, Ninette; Rechavi, Gideon; Kaminski, Naftali; Hochberg, Malka
CORPORATE SOURCE: Department of Dermatolology, The Hadassah-Hebrew University Medical Center, Jerusalem, Israel
SOURCE: Photodermatology, Photoimmunology & Photomedicine (2004), 20(3), 129-137
CODEN: PPPHEW; ISSN: 0905-4383
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background: Several recent studies have employed microarray profiling to study UVB-regulated gene expression in human skin. These studies are all based on UV-irradiated cultured cells that differ substantially from the intact tissues they are supposed to imitate. The purpose of the present study was to analyze the differential expression of UVB-regulated genes in intact human epidermis following in vivo UV irradiation Methods: The forearms of human volunteers were exposed to 4 MED of UVB in vivo, followed by removal of epidermal samples from exposed and non-exposed areas after 24

h. RNA samples were analyzed using oligonucleotide microarray (Affymetrix) technol. analyzing 12 500 genes simultaneously. Verification of selected genes was performed by semi-quant. reverse transcriptase polymerase chain reaction. Results: Gene expression patterns clearly distinguished UV-exposed epidermis from unexposed skin. Classification of these genes into functional categories revealed that several biol. processes are globally affected by UVB. Significant changes were seen in more than 800 genes. Conclusion: Human intact epidermis responds to a single low dose of in vivo UVB irradiation by differential regulation of numerous genes. Our results illustrate the power of global gene expression anal. of human epidermis to identify mol. pathways involved in UV-induced photodamage.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:737608 CAPLUS
DOCUMENT NUMBER: 139:240351
TITLE: Matrix metalloproteinase inhibitors in combination with hypothermia and/or radiotherapy for the treatment of cancer
INVENTOR(S): Nakajima, Motowo
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075959	A1	20030918	WO 2003-EP2365	20030307
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU, LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SE, SG, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW				
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
AU 2003214108	A1	20030922	AU 2003-214108	20030307
EP 1485131	A1	20041215	EP 2003-709764	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005526760	T	20050908	JP 2003-574232	20030307
US 2005232915	A1	20051020	US 2005-506936	20050606
PRIORITY APPLN. INFO.:			GB 2002-5537 A	20020308
			GB 2002-29054 A	20021212
			WO 2003-EP2365 W	20030307

OTHER SOURCE(S): MARPAT 139:240351
AB The invention provides a method of treating cancer in a subject in need of such treatment which comprises: radiotherapy, or cytotoxic therapy in combination with heat shock, and further comprises administering to the subject an effective amount of a matrix metalloproteinase inhibitor (Markush structures are included).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 20 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:521462 CAPLUS
DOCUMENT NUMBER: 137:88442

TITLE: Incensole and furanogermacrens and compounds in treatment for inhibiting neoplastic lesions and microorganisms
INVENTOR(S): Shanahan-Pendergast, Elisabeth
PATENT ASSIGNEE(S): Ire.
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002053138	A2	20020711	WO 2002-IE1	20020102
WO 2002053138	A3	20020919		
W: AE, AG, AT, AU, BB, BG, CA, CH, CN, CO, CU, CZ, LU, LV, MA, MD, UA, UG, US, VN, YU, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, AT, BE, CH, CY, DE, ES, FI, ML, MR, NE, SN, TD, TG				
AU 2002219472	A1	20020716	AU 2002-219472	20020102
EP 1351678	A2	20031015	EP 2002-727007	20020102
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004092583	A1	20040513	US 2004-250535	20040102
PRIORITY APPLN. INFO.:			IE 2001-2 A	20010102
			WO 2002-IE1 W	20020102

OTHER SOURCE(S): MARPAT 137:88442
AB The invention discloses the use of incensole and/or furanogermacrens, derivs. metabolites and precursors thereof in the treatment of neoplasia, particularly resistant neoplasia and immunodysregulatory disorders. These compds. can be administered alone or in combination with conventional chemotherapeutic, antiviral, antiparasite agents, radiation and/or surgery. Incensole and furanogermacren and their mixture showed antitumor activity against various human carcinomas and melanomas and antimicrobial activity against Staphylococcus aureus and Enterococcus faecalis.

L17 ANSWER 21 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:978584 CAPLUS
DOCUMENT NUMBER: 138:34125
TITLE: Determining changes in phenotype-specific gene expression in a cell by measuring changes in housekeeping and phenotype-specific gene expression
INVENTOR(S): Nishimura, Ichiro; Iida, Keisuke
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 21 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002197640	A1	20021226	US 2002-174658	20020619
WO 2004000867	A1	20031231	WO 2002-US19705	20020731
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,				

UA, UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002330858 A1 20040106 AU 2002-330858 20020731
PRIORITY APPLN. INFO.: US 2001-299910P P 20010621
US 2002-174658 A 20020619
WO 2002-US19705 W 20020731

AB The present invention provides an improved method for assessing, monitoring and/or determining the phenotype of cells and tissues. One aspect of the present invention is a method of fabricating phenotype specific gene (PSGs) and house keeping gene (HKGs) targets onto a microarray. Another aspect of the present invention provides a composition containing PSGs and HKGs as targets for high throughput assays including microarray analyses. Another aspect of the present invention is accessing, monitoring and/or determining the phenotype of tissue engineered cells derived from stem cells including embryonic stem cells, embryonic germ cells, fetal stem cells and adult stem cells by hybridizing cDNA probes to either PSG or HKG targets. These methods employ at least 25 PSG targets and no greater than 5000 HKG targets. Specific genes for use in measuring changes in given tissues are claimed.

L17 ANSWER 22 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:937303 CAPLUS
DOCUMENT NUMBER: 138:20443
TITLE: Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
INVENTOR(S): Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002355079	A	20021210	JP 2002-69354	20020313
PRIORITY APPLN. INFO.:			JP 2001-73183 A	20010314
			JP 2001-74993 A	20010315
			JP 2001-102519 A	20010330

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises preparing a nucleic acid sample containing mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample containing the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-β estradiol (E2), were found in mice by DNA chip anal.

L17 ANSWER 23 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights

reserved on STN DUPLICATE 3
ACCESSION NUMBER: 2003001967 EMBASE
TITLE: Infrared-A radiation-induced matrix metalloproteinase 1 expression is mediated through extracellular signal-regulated kinase 1/2 activation in human dermal fibroblasts.
AUTHOR: Schieke S.M.; Stege H.; Kurten V.; Grether-Beck S.; Sies H.; Krutmann J.
CORPORATE SOURCE: J. Krutmann, Inst. fur Umweltmedizinische Forsch., Heinrich-Heine-Universitat gGmbH, Auf'm Hennekamp 50, D-40225 Duesseldorf, Germany. krutmann@rz.uni-duesseldorf.de
SOURCE: Journal of Investigative Dermatology, (2002) Vol. 119, No. 6, pp. 1323-1329.
Refs: 41
ISSN: 0022-202X CODEN: JIDEAE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
014 Radiology
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Jan 2003
Last Updated on STN: 9 Jan 2003

AB In addition to ultraviolet radiation, human skin is exposed to infrared radiation from natural sunlight as well as artificial ultraviolet and infrared irradiation devices used for therapeutic or cosmetic purposes. The molecular consequences resulting from infrared exposure are virtually unknown. In this study we have investigated whether infrared has the capacity to affect gene expression in human skin cells. Exposure of cultured human dermal fibroblasts to infrared in the range of 760-1400 nm (infrared-A) induced the expression of matrix metalloproteinase 1 at the mRNA and protein level in a time- and concentration-dependent manner. Expression of tissue inhibitor of matrix metalloproteinase 1 remained unaltered. These effects were not mediated by the generation of heat by infrared-A. Furthermore, infrared-A did not induce heat shock protein 70 expression in human dermal fibroblasts under conditions that increased matrix metalloproteinase 1 expression. Here we provide evidence that infrared-A activated mitogen-activated protein kinase pathways. Extracellular signal-regulated kinase 1/2 and p38-mitogen-activated protein kinase were rapidly activated after infrared-A exposure. The mitogen-activated protein kinase/extracellular signal-regulated kinase inhibitor PD 98059, which specifically blocked the extracellular signal-regulated kinase pathway, prevented infrared-A-induced matrix metalloproteinase 1 expression. Upregulation of matrix metalloproteinase 1 expression by infrared-A was thus shown to be dependent on extracellular signal-regulated kinase 1/2 activation. In conclusion, this study demonstrates that infrared-A is capable of inducing matrix metalloproteinase 1 expression in human dermal fibroblasts via activation of the extracellular signal-regulated kinase 1/2 signaling pathway. This previously unrecognized property of infrared-A points to its possible role in the photoaging of human skin.

L17 ANSWER 24 OF 27 EMBASE
ACCESSION NUMBER: 2002087002 EMBASE
TITLE: New molecular targets of breast cancer therapy.
AUTHOR: Sauer G.; Deissler H.; Kurzeder C.; Kreienberg R.
CORPORATE SOURCE: Dr. G. Sauer, Department of Gynecology, University of Ulm

Medical School, Prittwitzstrasse 43, 89075 Ulm, Germany.
georg.sauer@medizin.uni-ulm.de
SOURCE: Strahlentherapie und Onkologie, (2002) Vol. 178, No. 3, pp. 123-133.
Refs: 131
ISSN: 0179-7158 CODEN: STONE4
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English; German
ENTRY DATE: Entered STN: 4 Apr 2002
Last Updated on STN: 4 Apr 2002

AB Background: The development of new chemotherapeutic agents and concepts of radiation therapy, administered as primary, adjuvant and palliative therapy, has led to new perspectives in breast cancer therapy. Apart from conventional chemotherapy, recently developed novel agents interfere with molecular mechanisms that are altered in cancer cells. Those targets are not necessarily breast cancer-specific. In this review we will focus on novel agents with potential or already proved benefit to breast cancer patients. Promising strategies include inhibition of growth factor receptors, blocking of tumor angiogenesis and signal transduction pathways, modulation of apoptosis, cancer vaccination, and inhibition of invasion and metastasis. Methods: Reports of relevant studies obtained from a search of MEDLINE and studies referenced in those reports were reviewed. Results: Apart from trastuzumab, other further developed compounds show promising results in clinical studies as a second generation of growth factor inhibitors. Different approaches in anti-angiogenetic therapy are under preclinical and clinical phase-II trials. Pro-apoptotic agents show synergistic effects with docetaxel in a clinical phase-I trial. Other compounds that target HSP 90, histone deacetylase and HMG-CoA reductase target atypical apoptotic pathways being lethal to tumor cells only but not to normal tissue, suggesting a tumor-specific way of action. MMP inhibitors have been demonstrating promising results in patients with refractory malignant pleural effusion in a phase-I trial. Several tyrosine kinase inhibitors currently under clinical investigation preliminarily show hopeful results in patients with advanced breast cancer. Furthermore, recent progress in defining the immunogenic epitopes of tumor antigens has rejuvenated the interest in cancer vaccines. Conclusion: Typical dose escalation studies leading to the highest clinically still tolerated dose do not appear to be equally appropriate for the estimation of efficiency of those compounds as for conventional cytotoxic regimes. Rather, escalation up to an amount of therapeutic agent that is sufficient for maximum target inhibition should be promoted, where classical measures of cytoreduction such as complete or partial remission are replaced both by time to progression and treatment failure as an appropriate measure of the efficacy of an agent.

ACCESSION NUMBER: 2005336848 EMBASE
TITLE: Focus on pancreas cancer.
AUTHOR: Jaffee E.M.; Hruban R.H.; Canto M.; Kern S.E.
CORPORATE SOURCE: E.M. Jaffee, Sidney Kimmel Cancer Center, Johns Hopkins University, Baltimore, MD 21231, United States.
ejaffee@jhmi.edu
SOURCE: Cancer Cell, (Jul 2002) Vol. 2, No. 1, pp. 25-28.
Refs: 27
ISSN: 1535-6108 CODEN: CCAECI
COUNTRY: United States

DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Sep 2005
Last Updated on STN: 1 Sep 2005

L17 ANSWER 26 OF 27 MEDLINE on STN
ACCESSION NUMBER: 2000144195 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10677564
TITLE: Induction of the putative protective protein ferritin by infrared radiation: implications in skin repair.
AUTHOR: Applegate L A; Scaletta C; Panizzon R; Frenk E; Hohlfeld P; Schwarzkopf S
CORPORATE SOURCE: Department of Obstetrics, Laboratory of Oxidative Stress and Aging, University Hospital, CHUV MAT-07, CH 1011 Lausanne, Switzerland.
SOURCE: International journal of molecular medicine, (2000 Mar) Vol. 5, No. 3, pp. 247-51.
Journal code: 9810955. ISSN: 1107-3756.
PUB. COUNTRY: Greece
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 13 Apr 2000
Last Updated on STN: 13 Apr 2000
Entered Medline: 7 Apr 2000
AB The modification of ferritin in human skin cells in vitro and in vivo following infrared-A irradiation by immunohistochemical analysis and ELISA were evaluated. In addition, we observed that IR-A is not capable of inducing frank damage to DNA (pyrimidine dimers, p53), induction of oxidative stress proteins (heme oxygenase, nitric oxide, superoxide dismutase, heat shock proteins) or proteases (collagenase, stromelysin, gelatinase) involved in carcinogenesis and photoaging of the skin. in vivo, basal levels of ferritin were heterogeneous for all individuals tested but all showed ferritin to stain precisely in the basal layer of unirradiated epidermis. Following IR-A radiation, the ferritin increase was localized to epidermal tissue and showed an increase from 120 to 220%. Parallel to the in vivo analysis, dermal fibroblasts were cultured from six individuals. Quantitative analysis for ferritin in cultured fibroblasts was assessed by ELISA and increases were seen to be dose-dependent and up to 130% of basal levels of ferritin following infrared-A. Our findings indicate that the putative defense system of ferritin that exists in human skin in vivo can be induced by infrared-A radiation and that these wavelengths may prove to be beneficial for human skin. Importantly, following the same doses of IR-A that induced ferritin levels, there was no alteration seen for nuclear DNA type damage, oxidative stress proteins or proteases involved in the degradation of skin. The increased concentrations of this antioxidant in human skin following acute UV radiation could afford increased protection against subsequent oxidative stress.

L17 ANSWER 27 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 4
ACCESSION NUMBER: 1992307017 EMBASE
TITLE: Changes in gene expression by 193- and 248-nm excimer laser radiation in cultured human fibroblasts.
AUTHOR: Rimoldi D.; Flessate D.M.; Samid D.

CORPORATE SOURCE: D. Samid, Department of Pathology, Uniformed Services Health Sci. Univ., Bethesda, MD 20814, United States
SOURCE: Radiation Research, (1992) Vol. 131, No. 3, pp. 325-331. ISSN: 0033-7587 CODEN: RAREAE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
022 Human Genetics
029 Clinical and Experimental Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 Nov 1992
Last Updated on STN: 8 Nov 1992

AB Tissue ablation by ultraviolet excimer lasers results in exposure of viable cells to subablative doses of radiation. To understand the potential biological consequences better, we have studied changes in gene expression in cultured human skin fibroblasts exposed to either 193- or 248-nm laser light. Northern blot analyses revealed that both treatments up-regulate a common set of genes, including interstitial collagenase, tissue inhibitor of metalloprotease, metallothionein, and the proto-oncogene c-fos. Dose-response and kinetic studies of collagenase induction by 193-nm radiation showed a maximal effect with 60 J/m(2) and at approximately 24 h. The induction was still persistent 96 h later. In addition to the commonly affected genes, known to be activated also by conventional UV light (254 nm) and tumor- promoting phorbol esters, other genes were found to be selectively induced by the 193-nm radiation. The heat-shock hsp70 mRNA, undetectable in controls and in cultures irradiated at 248 nm, was transiently induced 8 h after exposure to 193-nm radiation. Furthermore, a selective up-regulation of collagen type I expression was observed. The results indicate that the 193- and 248-nm radiations by excimer lasers elicit specific and different cellular responses, in addition to an overlapping pathway of gene activation common also to UV radiation by germicidal lamps. The laser-induced genes could serve as molecular markers in evaluating cell injury in situ.

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